Dissertation on

A STUDY ON THE EFFECTIVENESS OF EARLY SECTORAL PHOTOCOAGULATION IN BRANCH RETINAL VEIN OCCLUSION AT A TERTIARY CARE CENTER

M. S. OPHTHALMOLOGY

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MAY – 2019

CERTIFICATE

This is to certify that this dissertation titled **"A STUDY ON THE EFFECTIVENESS OF EARLY SECTORAL PHOTOCOAGULATION IN BRANCH RETINAL VEIN OCCLUSION AT A TERTIARY CARE CENTER"** is a bonafide record of the research work done by **Dr. DARA RESHMA.**., Post graduate in Regional Institute of Ophthalmology, Madras Medical College and Research Institute, Government General Hospital,Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2016-2019.

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Last but not the least, my heartful gratitude and sincere thanks to all my patients without whom this endeavor would not have been possible.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled, **"A STUDY ON THE EFFECTIVENESS OF EARLY SECTORAL PHOTOCOAGULATION IN BRANCH RETINAL VEIN OCCLUSION AT A TERTIARY CARE CENTER"** is a bonafide and genuine research work conducted by me under the guidance of **Prof. Dr.M.RAJAKUMARI, M.S., D.O.,** Head of the Department of uvea and retina services, Regional institute of ophthalmology & Government Ophthalmic hospital. Chennai-600008.

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CERTIFICATE OF APPROVAL

To
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Dear Dr.Dara Reshma,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON THE EFFECTIVENESS OF EARLY SECTORAL PHOTOCOAGULATION IN BRANCH RETINAL VEIN OCCLUSION AT A TERTIARY CARE CENTRE" - NO.10112017**

The following members of Ethics Committee were present in the meeting hold on **07.11.2017** conducted at Madras Medical College, Chennai 3

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We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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CONTENTS

ABBREVIATIONS

RVO – Retinal vein occlusion

ST BRVO – Supero – temporal branch retinal vein occlusion

IT BRVO – Infero – temporal branch retinal vein occlusion

CRVO – Central retinal vein occlusion

VEGF – Vascular endothelial growth factor

RPE – Retinal pigment epithelium

HDL – High density lipoprotein

OCT – Optical coherence tomography

FFA – Fundus fluorescein angiography

NVD – Neovascularization of disc

NVE – Neovascularization else where

NVI – Neovascularization of iris

NVA – Neovascularisation of angle

NVG - Neovascular glaucoma

IVTA – Intravitreal triamcinolone acetate

FDA – Food and drug administration

ICAM – Intercellular adhesion molecule

BCVA – Best corrected visual acuity

CNP – Capillary non perfusion

INTRODUCTION

RVO is an obstruction of retinal venous system, that involves either central vein or a branch retinal vein. RVO is typically due to the external compression or disease of the vessel wall.

RVO's are divided into

- Central retinal vein occlusion (CRVO)
- Hemi retinal vein occlusion (HRVO)
- Branch retinal vein occlusions (BRVO)
- Tributary vein occlusion
- Macular vein occlusion

BRVO is a venous occlusion at any branch of the central retinal vein. Occlusions occurring at the anterior part of the central retinal artery trunk results in a HRVO, which is consider as a subtype of either CRVO or BRVO.

The retina has a dual blood supply, with retinal vessels supplying the inner retina, and choroidal vessels supplying the outer retina extending from the outer part of inner nuclear layer .

The diagnosis of RVO is clinical and based on the fundus presentation like venous dilatation and tortuosity, flame shaped retinal hemorrhages, retinal edema, soft exudates affecting the sector of the retina drained by the affected vein in BRVO .

REVIEW OF LITERATURE

Occlusion of retinal blood vessels is a common occurrence which results in hypoxia of the retina. RVO is the second most common sight threatening retinal vascular disorder after diabetic retinopathy.

The global impact of RVO is significant , with an estimated 16.4 million adults affected worldwide . Large population based studies have shown that the incidence of RVO is 1.6 – 2.3 % over a period of 10 to 15 years , with a 15 year cumulative incidence of BRVO of 1.8 % and CRVO of 0.5 % .

The estimated prevalence of BRVO is 4.42 per 1000 persons and CRVO is 0.8 per 1000 persons . The prevalence increases by age and does not differ by gender .

The presence of open angle glaucoma is a risk factor for both BRVO and CRVO , with an odds ratio of 2.53 and 9.28 respectively .

Bilaterality is uncommon in both BRVO and CRVO , occurring in 6.3 % of eyes in 15 year Beaver Dam eye study cohort .

In 6.4 % of eyes after 5 years in the Blue mountains eye study cohort , the risk of vascular occlusion in fellow eye is estimated to be 0.9 % .

EMBRYOLOGY

The development of eyeball starts from around day 22 when the embryo has eight pairs of somites . The eye is formed from both ectoderm and mesenchyme .

The ectoderm that is derived from the neural tube gives rise to the retina , the nerve fibers of the optic nerve , and the smooth muscle of the iris .

The surface ectoderm on the side of the head forms the corneal and conjunctival epithelium , the lens , the lacrimal and tarsal glands .

The mesenchyme forms the corneal stroma , the sclera , the choroid , the iris , the ciliary musculature , part of vitreous body , and the cells lining the anterior chamber .

The endothelium of the cornea is believed to be of neural crest origin .

The rudimentary eyeball develops as an ectodermal diverticulum from the lateral aspect of the forebrain . The diverticulum grows out laterally towards the side of the head and becomes dilated to form the optic vesicle , while the proximal portion becomes constricted to form the optic stalk .

A small area of the ectoderm overlying the optic vesicle thickens to form the lens placode . The optic vesicle invaginates to form the double layered optic cup during 4^{th} week of gestation . The inferior edge of the optic cup is deficient , and this notch is

continuous with a groove on the inferior aspect of the optic stalk called the optic or choroidal or fetal fissure .

Vascular mesenchyme now grows into the optic fissure and takes with it the hyaloids artery . The optical fissure closes by 6^{th} week of gestation .The retina is developed from the two parts of the optic cup , the neurosensory retina from the inner wall and the retinal pigment epithelium from the outer wall .

NEUROSENSORY RETINA

The inner wall of the optic cup is a single layered epithelium with an internal and an external basement membrane .

By $4^{th} – 5^{th}$ week the primitive retina is arranged in two zones , they are the outer primitive zone (nuclear zone or germinal epithelium)and the inner marginal zone(layer of His) .

By $6^{th} – 7^{th}$ week the neuroepithelial cells divide by mitosis and differentiate into two layers , the inner and outer neuroblastic layers . These layers are separated by the transient fiber layer of chievitz .

The inner neuroblastic layer which further differentiates to form ganglion cells , muller cells , and amacrine cells . And the outer neuroblastic layer differentiates to form rods and cones , bipolar cells , and the horizontal cells .

DEVELOPMENT OF LAYERS OF SENSORY RETINA:

The nerve fiber layer becomes identifiable on the inner aspect of the inner neuroblastic layer .

The inner plexiform layer is identified by 10.5 weeks, there by obliterating the transient layer of chievitz .

The inner nuclear layer becomes identifiable in the posterior pole of retina and already contains the amacrine and muller cell bodies and shortly afterwards the bipolar and the horizontal cells differentiate from the outer neuroblastic layer and migrate into this new nucleated layer .

The outer nuclear layer is formed by the remaining components of the outer neuroblastic layer containing cell bodies of photoreceptors .

The outer plexiform layer is constituted by zone where fibers from this layer intermingle with those of the inner nuclear layer .

The external limiting membrane is identifiable in the early stages as rows of tight junctions between adjacent neuroblasts .

DIFFERENTIATION OF RETINAL LAYERS :

Differentiation of retinal layers starts by 6^{th} week of gestation and by 5 ½ months of gestation , all the layers of the adult retina are recognizable .

The macular area development is delayed upto 8^{th} month of gestation . Further differentiation of retina and macula continues until several months after birth .

Synaptogenesis in cone pedicle occurs approximately at 4 months and in rods occurs at 5 months . Photoreceptor outer segment formation commences at around 5^{th} month . Horizontal cells become distinguishable around the 5^{th} month .

Microglia , the resident tissue macrophages invade the retina via the retinal vasculature at 4months and invade the subretinal space from 10 weeks onwards .

Terminal expansions of the muller cells beneath the inner limiting membrane mature around 4.5 months , at around the same time as their processes can be identified between the rods and cones .

Cells of the outer wall of the optic cup become pigmented around 6^{th} week of gestation . Its posterior part forms the retinal pigment epithelium (RPE) of the retina and the anterior part continues forward in the ciliary body and iris as their pigmented epithelium .

Initially , the RPE comprises a mitotically active pseudostratified columnar ciliated epithelium . The cilia disappear as melanogenesis commences . The mitotic activity ceases by birth , thereafter growth of the eye and consequently of the RPE itself is accommodated by hypertrophy or enlargement of existing cells .

The mature RPE cells are hexagonal in shape , homogenous in size and in section appears as simple cuboidal epithelium . Melanin production in the pigment epithelial cells is gene – regulated .

RETINAL VESSELS

The fetal fissure along the optic stalk closes around the hyaloid artery , and the portion of the vessel within in stalk becomes the central retinal artery . A branch of the primitive maxillary vein located within the optic stalk is the likely precursor of the central retinal vein .

Early in the 4^{th} month of development , primitive retinal vessels emerge from the hyaloids artery near the optic disc and enter the developing nerve fiber layer .

Signals from bimolecular agents guide the growth and pathway of neurons and likely also guide the growth of these retinal vessels . The vessels of the retina continues to develop forming arterioles , venules , and capillary beds , but all vessel structure is not completed until approximately 3 months after birth , with the vessels to the nasal periphery completed before those of temporal periphery .

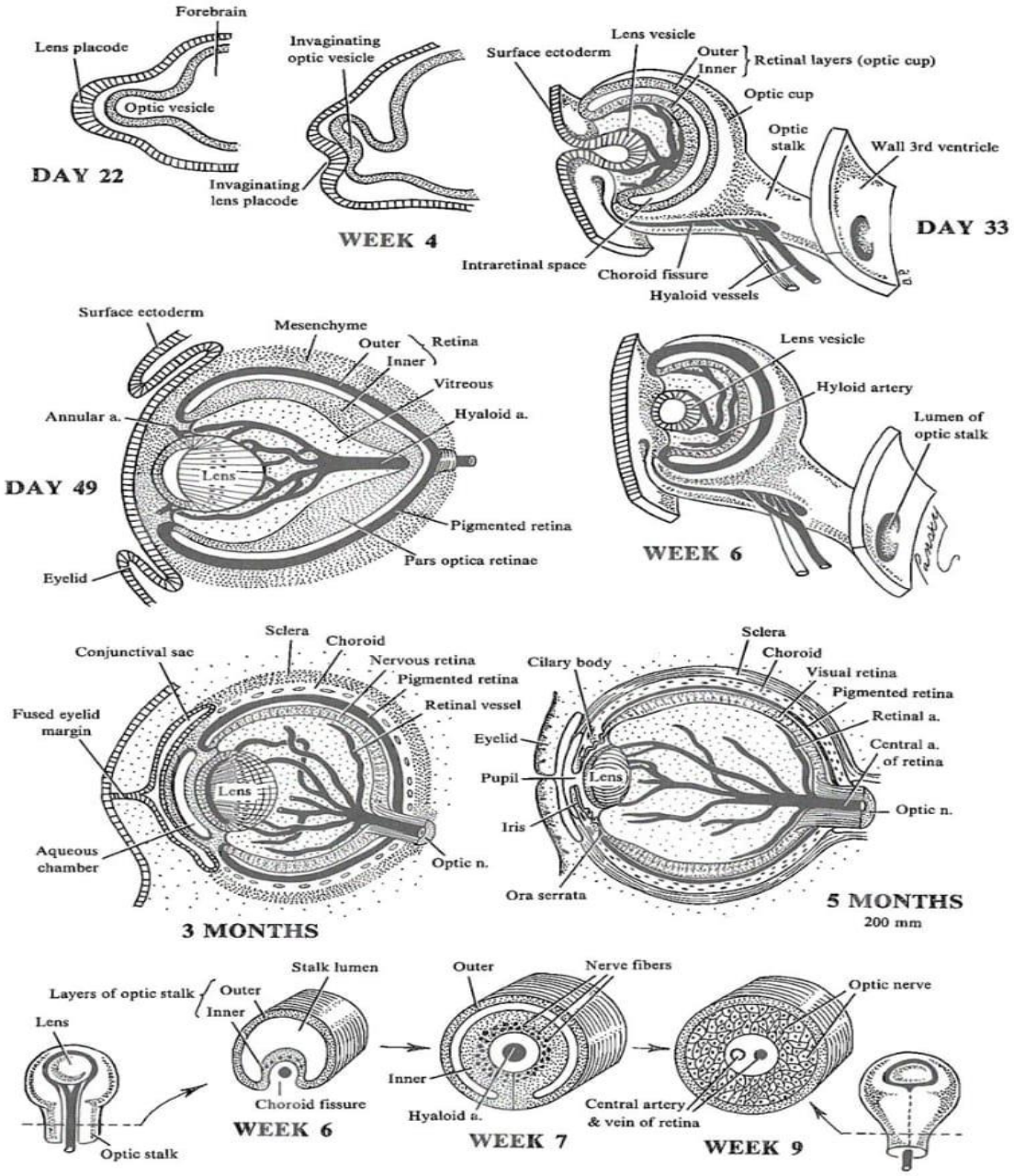

Fig 1 : Embryology of the eye

ANATOMY

Retina is the innermost layer of the eyeball. It is a thin, delicate and transparent membrane. It is the most highly developed tissue of the eyeball.

GROSS ANATOMY :

Retina extends from optic disc to the ora serrata . Thickness of retina at the posterior pole in the peripaillary region is approximately 0.56mm, at the equator 0.18 to 0.2mm, and at the ora serrata approximately 0.1mm. It appears purplish red due to visual purple of rods.

Grossly, on ophthalmoscopic examination, fundus can be divided into three distinct regions.

- Optic disc
- Macula lutea
- Peripheral retina

OPTIC DISC

It is a pale-pink , well defined circular area of about 1.5mm diameter. At the optic disc, all the retinal layers terminate except the nerve fibres, which pass through the lamina cribrosa to run into the optic nerve.

Physiological cup of the optic disc is a depression seen in it. The central retinal vessels emerge through the centre of this cup. It varies in size, shape, position and depth in different individuals.

MACULA LUTEA

It is a yellow spot (because of oxygenated carotenoids) of about 5.5mm in diameter , situated at the posterior pole of the eyeball . Histologically it is the region with more than 1 layer of ganglionic cell nuclei . It comprises of 3 main areas .

- **Fovea**- It is the central depressed part of the macula. It is about 1.50mm in diameter and 1.55mm in thickness . It corresponds to 5^0 of the visual field and is the most sensitive part of the retina.

  Margo fovea - Margin of fovea is a ring-like reflection of the internal limiting membrane (microscopically) .

  Foveola - Forms the central floor of the fovea. It is situated about 2 disc diameter away from the temporal edge of the optic disc . It is 0.35mm in diameter .

Umbo - Is a tiny depression in the centre of foveola which corresponds to the visible foveolar reflex seen in normal individuals. Loss of this reflex is an early sign of damage. Greatest concentration of cones is found in umbo . Its diameter is about 150 – 200 µm

Clivus - Extends from the margin of fovea to the margin of foveola. It consists of the peripheral vascular and central foveolar avascular zone (FAZ) .

FAZ is located inside fovea but outside foveola .

- **Parafovea-** It surrounds the foveolar margin. Histologically it is characterised by 4-6 layers of ganglion cells and 7-11 layers of bipolar cells .

- **Perifovea-** It measures 1.5mm in width and surrounds the parafoveolar area. It is characterized by several layers of ganglionic cells and six layers of bipolar cells.

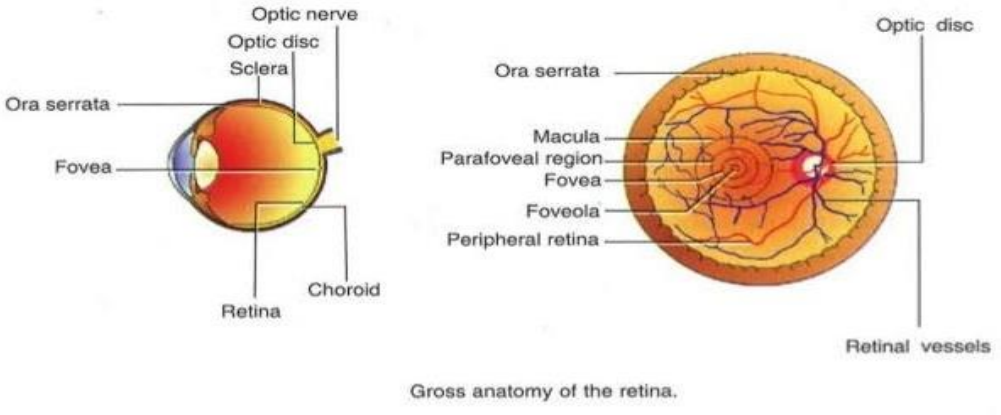

Fig 2: Gross anatomy of the retina .

PERIPHERAL RETINA

It is divided into four regions

- ⦿ **Near periphery**- It is a region of about 1.5mm width around the macula lutea.
- ⦿ **Mid-periphery**- It is a 3mm wide zone around the near periphery. Its outer limit corresponds to the equator.
- ⦿ **Far periphery**- It extends from equator to the ora serrata. Its width varies depending on the ocular size and refractive error.

- The average circumference of the eye is 72mm at equator and 60mm at the ora serrata. The average width of far periphery is about 6mm.
- As the peripheral retinal pathologies are charted in clock hours, 1-clock hour corresponds to 5-6mm of far periphery. Thus, the belt of peripheral retina can be divided into 12 squares.

⦿ **Extreme periphery**- Area of ora serrata and pars plana.

- ***Ora serrata***- It is the peripheral margin where the retina ends and ciliary body starts. At the ora, the sensory retina is firmly attached both to the vitreous and retinal pigment epithelium. Its distance from the limbus is 6mm nasally and 7mm temporally. It is a water shed zone between anterior and posterior vascular systems. So, peripheral retinal degenerations are more common.

MICROSCOPIC STRUCTURE OF THE RETINA

It consists of ten layers from two distinct functional components, the pigment epithelium and the sensory retina.

1. Retinal pigment epithelium
2. Layer of rods and cones
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Innernuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Nerve fibre layer
10. Internal limiting membrane

1) RETINAL PIGMENT EPITHELIUM (RPE)

It is the outer most layer of retina. It consists of single layer of hexagonal-shaped cells containing pigment. It is firmly adherent to the underlying Bruch's membrane (basal lamina of choroid) and loosely attached to the layer of rods and cones of the sensory retina.

The space between RPE and the sensory retina is called sub-retinal space. A separation of the RPE from the sensory retina is called retinal detachment and the fluid between the two layers is called subretinal fluid.

Electron microscopy shows that adjacent RPE are connected with each other by tight junctions and constitute outer retinal barrier. In cross-section it can be differentiated into apical and basal configurations .

RPE plays important role in photoreceptor renewal and recycling of vitamin A, actively pumps ions and water out of subretinal space, transport of nutrients and metabolites, phagocytic action, regenerative and repairative function, manufactures pigment and electrical homeostasis.

2) LAYER OF RODS AND CONES

They are the end organs of vision which transform light energy into visual impulse. Rods contain substance called rhodopsin and serve for the peripheral vision. Cones are responsible for central vision . Number of photoreceptors are about 120 million rods and 6.5 million cones .

3) EXTERNAL LIMITING MEMBRANE

It is a membrane extending from the ora serrata to the edge of the optic disc . Processes of rods and cones pass through this . It is formed by the junctions between photoreceptors and muller cells .

4) OUTER NUCLEAR LAYER

It is formed by the nuclei of rods and cones . Cone nuclei are larger than rod nuclei . Rod nuclei form bulk of this multilayered outer nuclear layer except in the cone dominated foveal region .

5) OUTER PLEXIFORM LAYER

It contains the synapses between the rods and cones with the dendrites of the bipolar cells. It marks the junction of the end organs of vision and first order neurons in the retina**.** Consists of oblique fibers that are deviated from fovea , also called as henle's layer .

6) INNER NUCLEAR LAYER

This layer disappears at fovea and in rest of the retina consists of bipolar cells, horizontal cells, amacrine cells, the soma of the muller's cells and the capillaries of the retinal vessels. The bipolar cells are neurons of first order of vision.

7) INNER PLEXIFORM LAYER

This layer consists of synapses between the axons of bipolar cells, dendrites of the ganglion cells and the processes of amacrine cells. This layer is absent at the foveola.

8) GANGLION CELL LAYER

The cell bodies and the nuclei of the ganglion cells lie in this layer. It is composed of single row of cells, except in macular region where it is multi layered. It is absent in the foveola. They are variously classified as W, X and Y ganglion cells, Off centre and ON centre ganglion cells, P and M ganglion cells, Monosynaptic and polysynaptic ganglion cells .

9) NERVE FIBER LAYER

It consists of the unmyelinated axons of the ganglion cells which cover the optic nerve head, pass through lamina cribrosa and become unsheathed by myelin posterior to lamina .

Fibres from the nasal half of the retina come directly to the optic disc as superior and inferior radiating fibres.

Fibres from the macular region pass straight in the temporal part of the disc as papillomacular bundle (pmb).

Fibres from the temporal retina arch above and below the macular and papillamacular bundle as superior and inferior arcuate fibres.

Arcuate fibres which occupy the superior and inferior temporal quadrants are most sensitive to glaucomatous damage .

Macular fibres which occupy the lateral quadrant are most resistant to glaucomatous damage , therefore explains the preservation of central vision till the end .

10) INTERNAL LIMITING MEMBRANE

It mainly consists of PAS positive basement membrane that forms the interface between retina and vitreous. It consists of collagen fibrils, proteoglycans , basement membrane and plasma membrane of the muller cells.

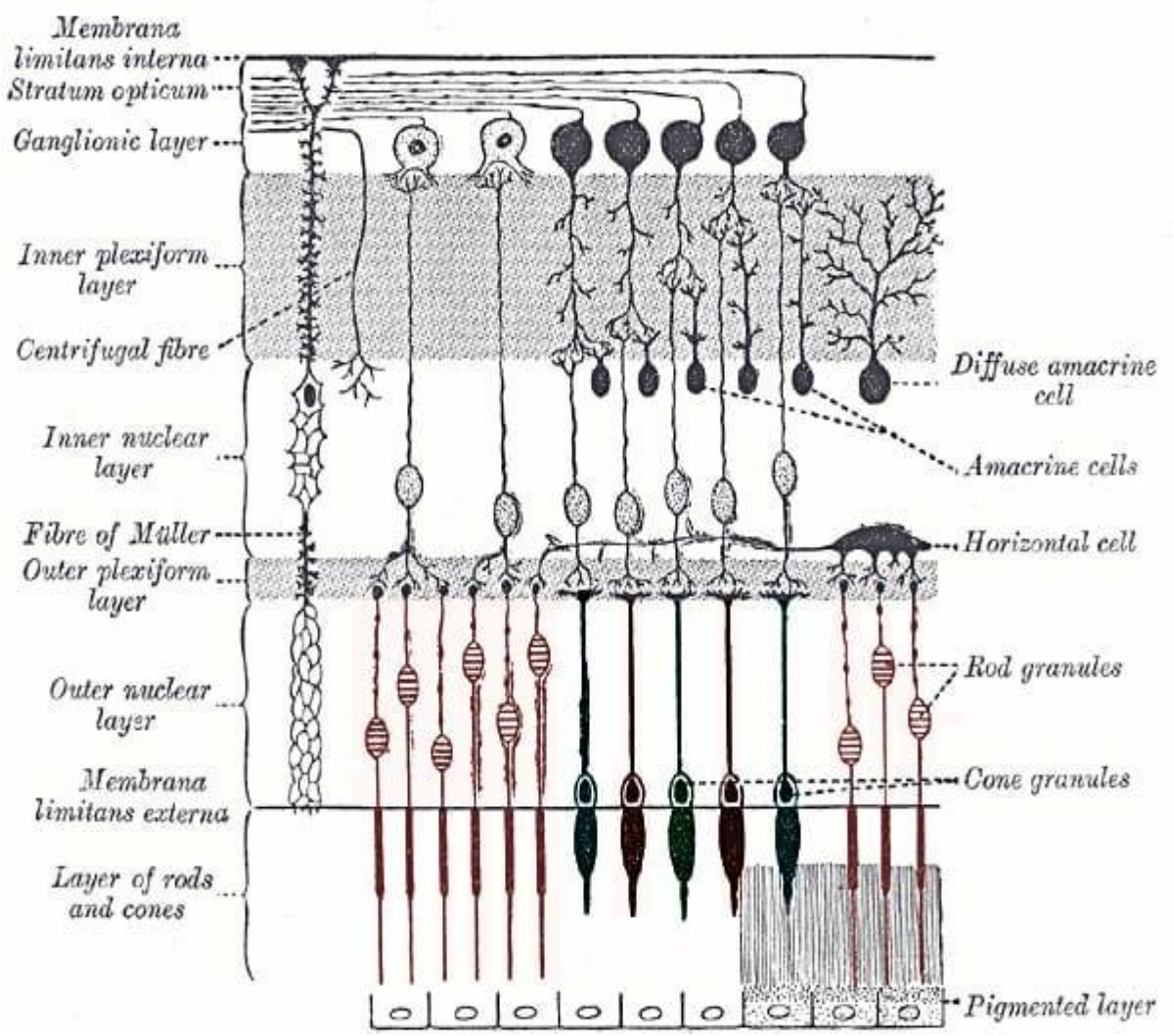

Fig 3 : Layers of the Retina

BLOOD SUPPLY OF THE RETINA

- Outer four layers of the retina get their nutrition from the choriocapillaries.
- Inner six layers of the retina get their supply from central retinal artery.
- Outer plexiform layer gets its blood supply partly from central retinal artery and partly from choriocapillaries by diffusion.
- Macular region gets its blood supply from the superior and inferior temporal branches of central retinal artery. Sometimes, cilioretinal artery(a branch of ciliary vessels) is seen originating in a hook shaped manner within the temporal margin of the disc. It runs towards the macula and supplies it and helps retain the central vision in the event of central artery occlusion.
- Retinal vessels are end arteries. Anastomosis between the retinal arteries and ciliary vessels does exist with the vessels which enter the optic nerve head from the arterial circle of zinn or haller, This arterial circle is formed by an anstomosis between 2 and 4 short posterior ciliary arteries and lies in the sclera around the optic nerve.

Central retinal artery

The first branch of ophthalmic artery, arises near the optic foramen and courses ahead with 5-6 right angle bends.

- Outside the optic nerve- It runs below the optic nerve adherent to the dural sheath to about 10-15mm behind the eyeball, where it bends upwards to pierce the dura and arachnoid.

- In the subarachnoid space - It bends forwards and upwards at nearly right angle and invaginates the pia to reach the centre of the nerve .

- In the center of the nerve - It bends forward and then in company with the vein, which lies on the temporal side, it passes anteriorly and pierces lamina cribrosa to appear inside the eye.

- In the optic nerve head - It lies superficially in the nasal part of physiology cup, where it divides into two branches superior and an inferior, each of which divides into a temporal and nasal branch.

- In the retina – The four terminal branches of central retinal artery divide dichotomously as they proceed towards the ora serrata, where they end without anastomosis.

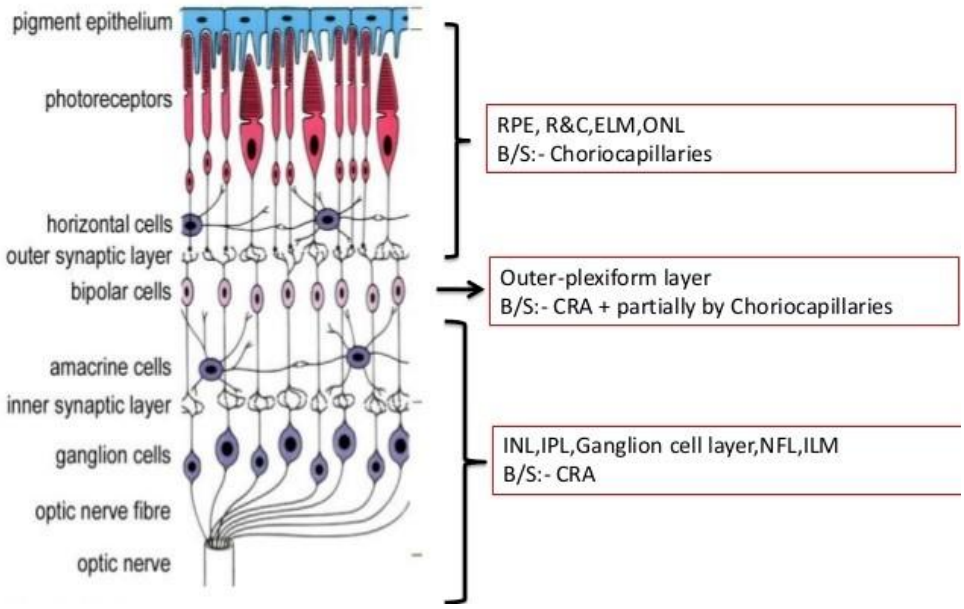

Fig 4 : Blood supply of retina

ARRANGEMENT OF RETINAL CAPILLARIES

The teriminal fundus arterioles bend sharply and almost dip vertically into the retina . In most of the extramacular fundus , there are two capillary networks , a superficial capillary network which lies at the nerve fiber layer and a deep capillary network that lies between the inner nuclear layer and the outer plexiform .

Peripherally the capillary network is reduced to a single layer as the ora serrata is approached .

In the parafoveal zone the capillary network is three layered . There exists a capillary – free zone in the fovea of about 500µm in diameter called the foveal avascular zone .

In the peripapillary region , the capillary network becomes four layered to support the extremely thick nerve fiber layer .

BLOOD – RETINAL BARRIER

The endothelial cells of the normal retinal capillaries are bound together by intercellular junctions of zonula occludens type . These junctions prohibit flow of fluid and solutes from the vascular lumen into the retinal interstitium and thus form a blood retinal barrier .

The endothelial cells are surrounded by a layer basement membrane followed by a layer of pericytes which is inturn surrounded by a layer of basement membrane . The endothelial cells and pericytes are in 1:1 ratio normally in young individuals .

PHYSIOLOGY

RETINAL METABOLISM

The respiratory rate of retina is twice that of the brain . Half if it accounted to the ellipsoidal region which is rich in mitochondria . The retina does not require insulin for glucose to enter the cells . Muller cells contain glucose - 6 – phosphatase activity which release glucose from their stores into neuro retina .

Retina ceases its function if blood supply is stoped for few minutes . Therefore an adequate oxygen supply , glucose , lipids , aminoacids , vitamins , minerals are all needed for retina to function properly .

Retina require a minimum of 30mg/100ml of carbohydrates to function normally . Deprivation of glucose for 8 to 10 minutes results in irreversible cell damage .

Carbohydrate breakdown is mainly by glycolysis to pyruvate and lactate . Secondly by krebs cycle to carbonic acid and water . In retina glycolysis occurs even when there is sufficient oxygen supply unlike other tissues .

Glycogen is stored in glial cells such as muller fibers . They act as buffer against changes in the concentration of glucose .

BRANCH RETINAL VEIN OCCLUSION

BRVO is a common retinal vascular disorder of the elderly. The Beaver Dam Study estimated the 15 year cumulative incidence of RVO at 2.3% in population , majority of these are BRVO (78%) .

BRVOs occur approximately three times more commonly than CRVO's . Men and women are affected equally , with the usual age of onset between 60 and 70 years .

Visual loss from a branch retinal vein occlusion usually is caused by macular edema , macular ischemia or vitreous hemorrhage .The common compliants of BRVO patients are sudden onset of blurring of vison or visual field defect .

Systemic diseases such as hypertension and atherosclerosis are risk factors for BRVO, because they lead to thickening of the retinal arteries . Other risk factors are diabetes , smoking , obesity , hyperlipidemia , glaucoma .

Rarely, local ocular diseases, especially of an inflammatory nature can result in secondary BRVO. RVO is associated with immunological diseases in young patients .

It has been reported in diseases such as toxoplasmosis , Eales disease , behcet's disease , ocular sarcoidosis , Macroaneurysms , Coats disease , retinal capillary hemangioma , and optic disc drusen are linked to BRVO .

Elevated plasma homocysteine , antiphospholipid antibodies , low serum folate levels , shorter axial lengths are also associated with raised risk of vein occlusion .

High serum HDL levels and light to moderate alcohol consumption are associated with decreased risk of vein occlusion

PATHOGENESIS :

Most epidemiological and histopathological evidence implicates arteriolar disease as the underlying pathogenesis . Open angle glaucoma is the most frequent local alteration predisposing to RVO , as it compromises the venous outflow by increasing IOP .

Branch retinal vein obstruction almost always occurs at an arteriovenous crossing , where the artery and vein share a common adventitial sheath . The artery nearly always is anterior (innermost) to the vein .It is postulated that a rigid, arteriosclerotic artery compresses the retinal vein , which results in turbulent blood flow and endothelial damage , followed by thrombosis and obstruction of the vein .

In shorter wave length eyes vitreous may also cause compression at the arteriovenous crossing sites and increase the risk of BRVO .

Most BRVOs occur superotemporally , probably because this is where the highest concentration of arteriovenous crossings lies . BRVO occurs superotemporally 52.3% of the times , inferotemporally 38.5% of the times , nasal quadrant 9.2% of the times .

Nasal quadrant BRVO's are likely under represented as most patients are asymptomatic and thus do not seek ophthalmic help .

The natural history of RVO is highly variable , in some cases the retinal findings progressively disappear and visual outcome is good , while other cases end up with severe complications like neovascularization , vitreous hemorrhage , NVG , macular edema .

In BRVO 5 – 15% of eyes develop macular edema over the first year . And vitreous hemorrhage develops in 40% of eyes with BRVO within 9months of presentation .

Neovascularization in vascular occlusion occurs due to tissue ischemia leading to elevation of angiogenic factors is thought to be the major important factor in neovascularization after RVO .

ANGIOGENIC FACTORS :

VEGF also known as VEGF A or VEGF 1 is a heparin – binding dimeric glycoprotein with disulfide – linked subunits , which share significant sequence homology with the A and B chains of PDGF .

VEGF 165 is the predominantly expressed isoform , it is the critical isoform for both development and pathological retinal angiogenesis .

Other endothelial growth factors are VEGF B , VEGF C or VEGF 2 , VEGF D , VEGF E . VEGF has shown to induce the expression of plasminogen activation in microvascular endothelial cells , which is important in the extracellular proteolysis necessary for capillary formation .

VEGF also induces the expression of endothelial cell $\alpha 1\beta 1$ and $\alpha 2\beta 1$ integrins , that are important for migration . VEGF upregulates endothelial cell fenestrations in choroidal plexus and choroidal capillaries .

Leucocyte adhesion is important in vascular leakage , VEGF increases the expression of ICAM – 1 on endothelial cells resulting in increased leukostasis , which mediates the break down of blood retinal barrier .

CLINICAL FEATURES

Usually the patients present with symptoms of sudden painless loss of vision or a visual field defect .

On examination the fundus shows a wedge shaped intraretinal hemorrhages with the apex at the site of blockage . These hemorrhages are more marked if occlusion is of ischemic type and less marked if the occlusion is of nonischemic type .

The intraretinal hemorrhages distribution is determined by the location of the venous blockage . If the block is at the optic nerve head two quadrants are affected , if the block is peripheral to disc one quadrant or less is involved , if the block is peripheral to the tributary veins draining the macula then macula is spared and there will be no drop in vision .

As the time progresses the intraretinal hemorrhage may be absorbed but the vascular abnormalities that occurred during the acute phase will persist like the capillary nonperfusion , dilation of the capillaries , microaneurysms , telangectatic vessels , collateral vessels . Macular edema may be present in some cases which can be quantified by OCT .

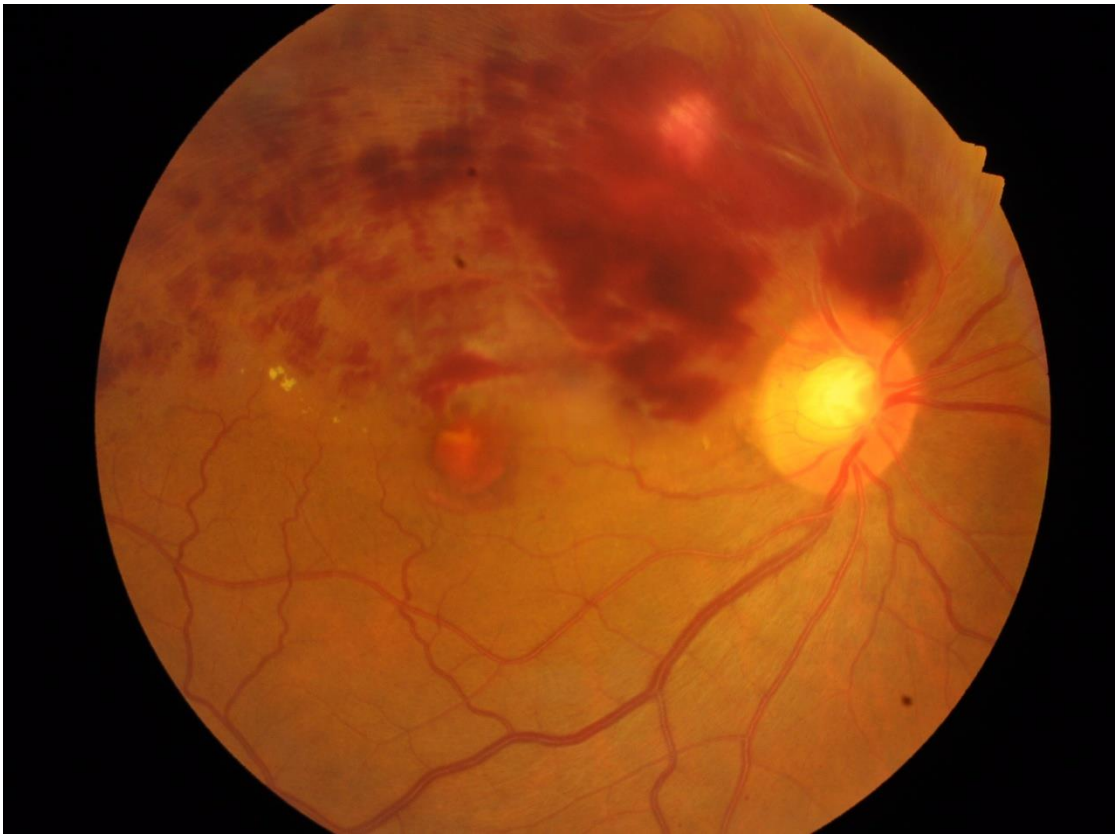

Fig 5 : Fundus photograph of a right eye showing ST BRVO with wedge shaped hemorrhages .

COMPLICATIONS

The most common complications of BRVO are macular edema , macular ischemia , and neovascularization . In acute phase the intraretinal hemorrhages can obscure the assessment of the macular edema or macular ischemia status , since hemorrhages produce a blocked fluorescence on FFA . Therefore OCT is an important ancillary test to look for macular edema .

Collaterals crossing the horizontal raphe are a salient feature of BRVO and their presence should prompt consideration of a remote BRVO .

Epiretinal membrane , NVE , NVD , NVI , NVG , vitreous hemorrhage , tractional retinal detachment are late complications due to ischemia . Except for macular ischemia these complications can be prevented or treated in most cases . So it is very important to follow a BRVO patient periodically.

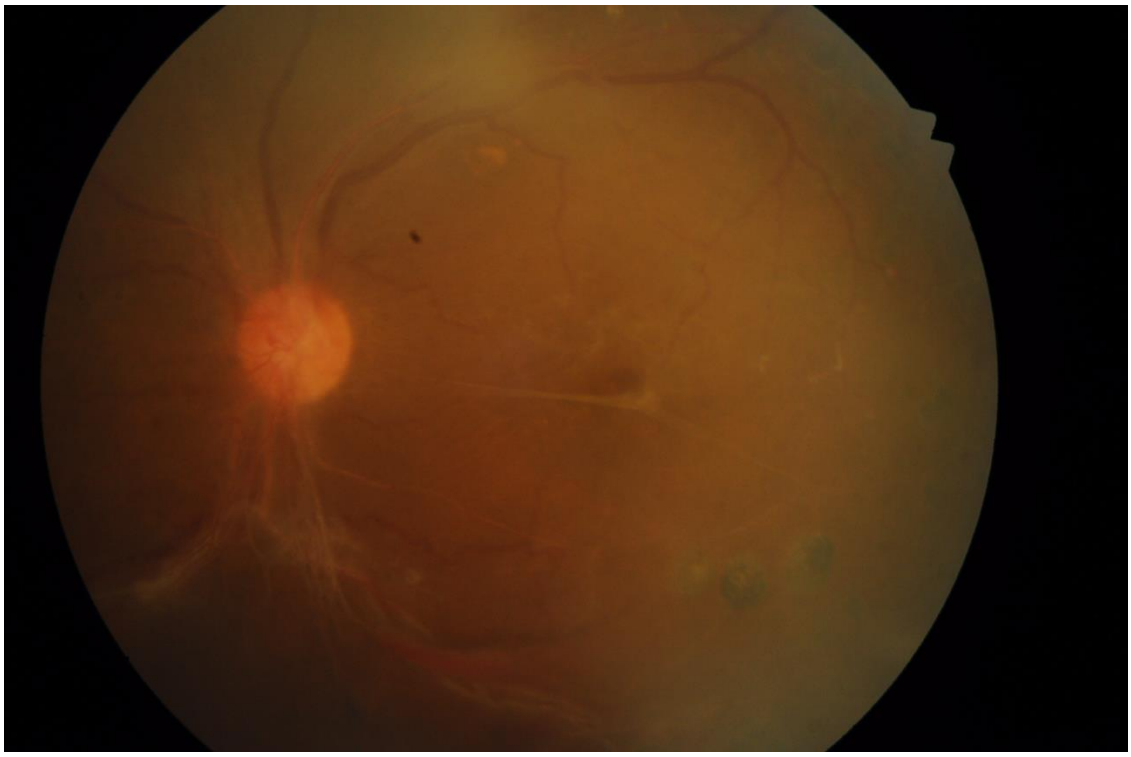

Fig 6: A fundus photograph of a left eye showing NVD with fibrovascular proliferation in an IT BRVO patient .

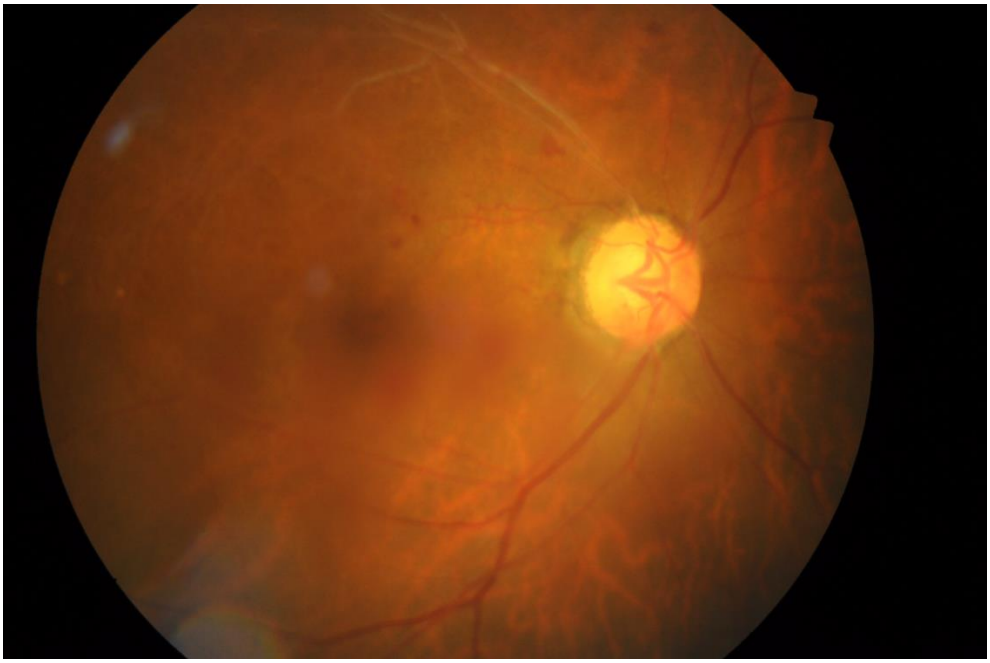

Fig 7a: A fundus photograph of a right eye showing ST BRVO , with NVE and NVD`

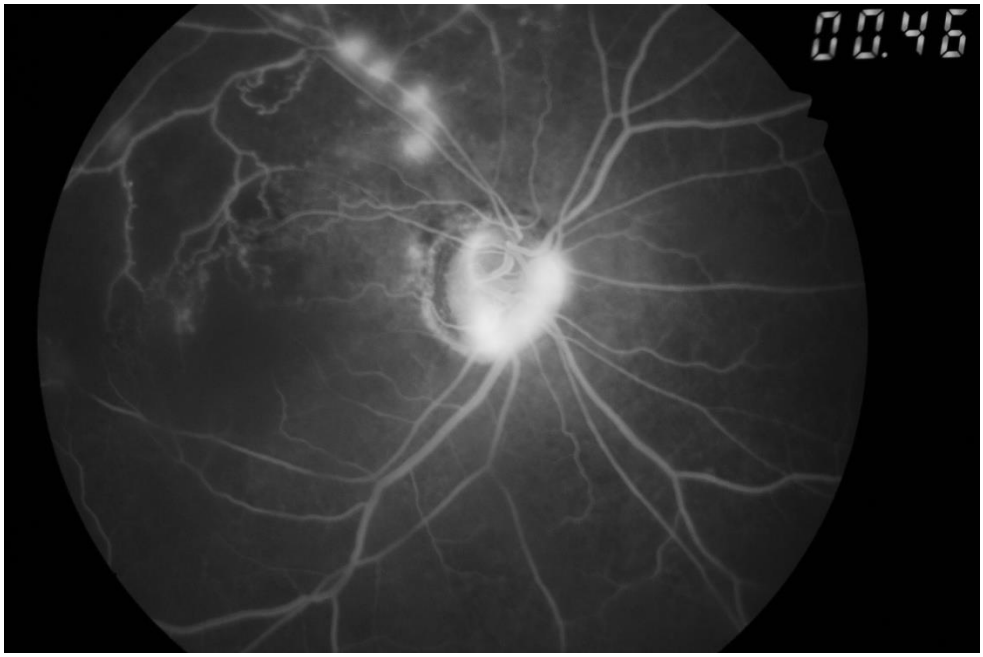

Fig 7b: FFA picture of the above patient showing early hyperfluorescence over the disc and in the superior arcade region suggestive of NVD and NVE respectively .

CLINICAL EVALUATION

The clinical examination should include complete ophthalmic examination . Glaucoma and inflammatory diseases should be ruled out as they are associated with BRVO . In chronic cases careful iris examination and angle examination should be done to look for rubeosis or NVA .

FUNDUS FLUORESCEIN ANGIOGRAPHY

FFA helps to understand the vascular characteristics that have prognostic significance like macular leakage , macular edema , macular ischemia , capillary non-perfusion areas . FFA is the only technique that accurately defines the capillary abnormality in BRVO .

The characteristic findings on FFA in BRVO are delayed filling of the blocked vessel , blocked fluorescence due to intra retinal hemorrhages , capillary nonperfusion areas , microanuerysms , telangiectatic collateral vessels may cross the horizontal raphe , vessel wall staining near the site of blockage , dye extravasation from macular edema or neovascularization .

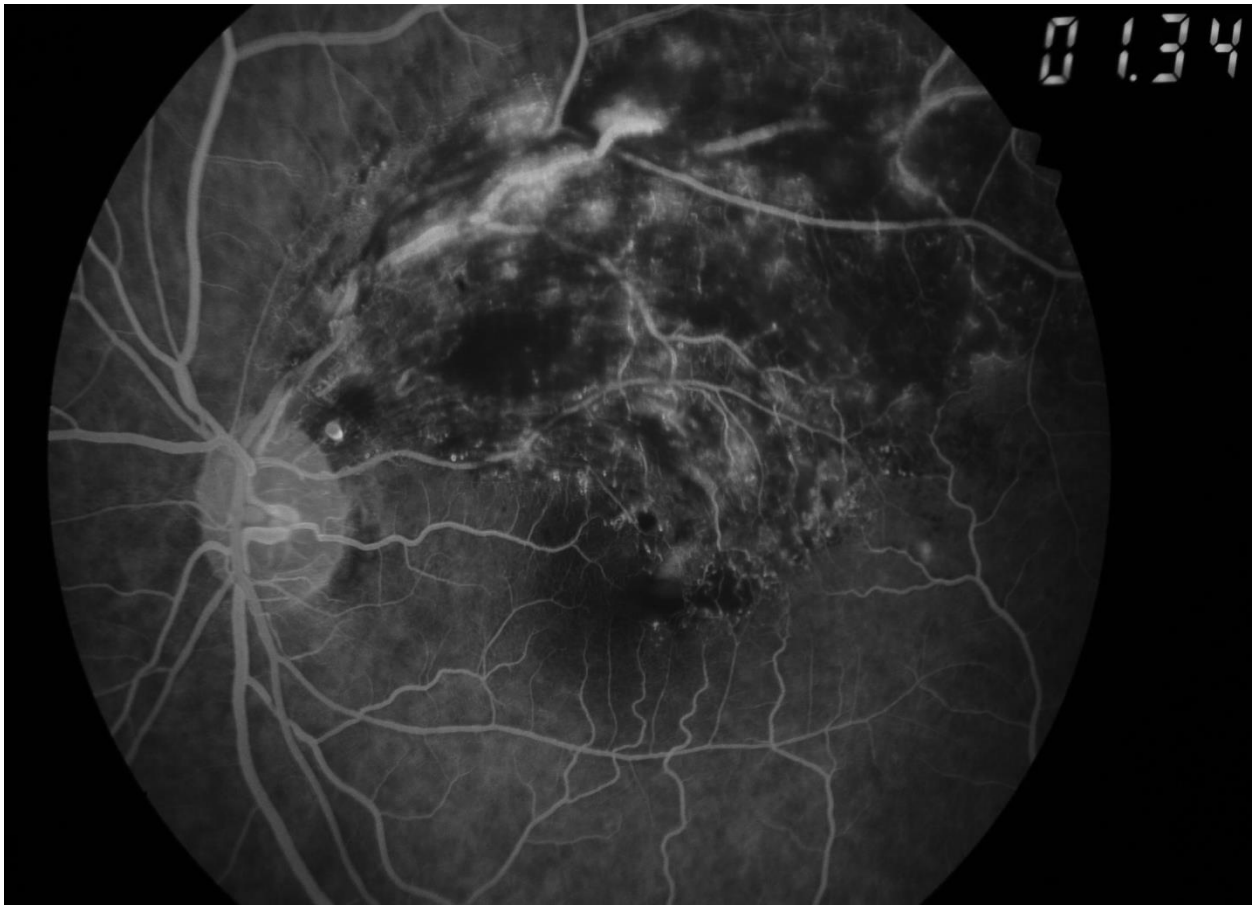

Fig 8 : A FFA picture showing left eye ST BRVO with blocked fluorescence due to intaretinal hemorrhages and hyperfluorescence along the vessel wall due to vessel wall staining .

WIDE – FIELD ANGIOGRAPHY

Recently a Ultrawide field angiography using the Optos C200MA revealed a correlation between nonperfusion in the peripheral retina with macular edema and neovascularisation . But wide field angiography is not frequently used in BRVO .

OPTICAL COHERENCE TOMOGRAPHY

OCT is a rapid and noninvasive method to quantify macular edema . It is extremely useful in acute phase when FFA cannot give much information regarding the macular edema due to intraretinal hemorrhages . OCT is used to monitor the treatment response of macular edema .

The characteristic findings of BRVO on OCT are cystoid macular edema , intraretinal hyperreflectivity due to hemorrhages , shadowing from edema and hemorrhages , subretinal fluid , Photoreceeptor inner segment and outer segment junction abnormalities from long standing macular edema and ischemia in chronic cases .

DIAGNOSTIC WORK UP

BRVO usually occurs in old age , but if presents in young age underlying predisposing factors should be evaluated . Any systemic illness or medication history , if in females oral contraceptive use history should be obtained as it may lead to hypercoagulable states .

Patient should be screened for hypertension , diabetes . In suspected patients , infectious disease like Lymes disease , syphilis , human immunodeficiency virus .

In suspicion of inflammatory diseases or coagulopathies , complete blood count , prothrombin time / partial thromboplastin time ,international normalized ratio , lipid profile , serum homocysteine , anticardiolipin antibodies , antinuclear antibodies , with lupus anticoagulant , protein C and S , activated protein C resistance (factor V Leiden)

In patients older than 60 years additional workup is not needed as these cases are usually idiopathic or due to hypertension or atherosclerosis

In cases of bilateral or multiple BRVO's search for a infectious or inflammatory disorder or a hypercoagulopathy may be warranted .

DIFFERENTIAL DIAGNOSIS :

Differential diagnosis of BRVO include ,

- Hypertensive retinopathy
- Diabetic retinopathy
- Radiation retinopathy
- Macular telangiectasia
- Retinal angiomatous proliferation

TREATMENT

MEDICAL TREATMENT

Treatment of the underlying disease is important . Treatment of the BRVO is focused on the management of vision – limiting complications .

LASER TREATMENT

Laser treatment in BRVO is done according to the BVOS . BVOS is a multicenter randomized clinical trail supported by the National Eye Institute , which began in 1977 . It states that argon laser photocoagulation may reduce the visual loss from macular edema in patients who meet the eligibity criteria of the study .

The eligibility criteria include fluorescein proven perfused macular edema involving foveal center , absorption of intraretinal hemorrhages from foveal center , recent BRVO (3 – 18 months duration) , no diabetic retinopathy , vision reduced to 20/40 or worse after best refraction .

According to BVOS Argon laser photocoagulation is given in grid pattern to all the leaking areas on FFA . Coagulation extended not closer to the fovea than the edge of the capillary free zone and not further into the periphery than the major vascular arcade .

Treatment parameters are , a spot size of 100µm diameter , for a duration of 0.1 second and power setting sufficient to produce a medium white burn .

FFA can be repeated after 2 – 4 months and augmentation of photocoagulation can be done to the residual areas of leakage if reduced visual acuity persists . When treated in this manner treated eyes showed two or more snellen lines improvement than untreated eyes in the consecutive visits .

After 3 years of follow up 63% of treated eyes showed two or more lines improvement in vision , compared to 36% of untreated eyes , The average gain in visual acuity for treated eyes was one more snellen line than in untreated eyes .

It is important to do a high quality FFA before laser photocoagulation . It is also important to follow the patient for a period of time to ascertain that the macular edema is not resolving spontaneously . During this period of follow up , it should be demonstrated that the intraretinal hemorrhages are clearing and the foveal center is devoid of any hemorrhages that could be the cause of the decreased vision .

The FFA must demonstrate that the macular edema involves the center of the fovea and that there is no large capillary non perfusion areas adjacent to the capillary free zone that could explain the visual loss .

In grid photocoagulation the absorption takes place at the RPE level . The laser is not aimed at closing the leaking and dilated capillary vessel . It is not understood how the laser helps in decreasing the macular edema , one reason could be the grid patter coagulation causes thinning of the outer retina decreasing the oxygen demand and

increasing the choroidal delivery of oxygen to inner retina , producing a autoregulatory constriction of the retinal vessels in the leaking area .

It is important to clearly define the landmarks before application of grid laser to avoid foveal burns . If the landmarks are masked , like in most cases of BRVO , the laser should be given well periphery from the capillary free zone . In BVOS for grid photocoagulation , the laser used is the Argon blue – green wavelength laser .

If macular edema still persists in further visits , the laser can be placed more closer to the capillary free zone than the previous laser treatment . Performing grid laser in this repetitively staged manner is much safer and just as effective as the single treatment . It has never been established that macular edema must be treated immediately or persistent macular edema causes irreversible macular damage in first 2 – 3 years .

The summary of BVOS recommendation for treatment of acute BRVO is waiting atleast 3 – 6 months before considering laser therapy . If vision is reduced to less than 20/40 or worse , wait for 3 – 6 months and a high quality FFA should be done after clearing of the intraretinal hemorrhages to evaluate the macular edema and macular ischemia . If macula is perfused and the vision loss is 20/40 or worse without spontaneous improvement then grid laser can be considered . If macular ischemia is the cause for vision loss laser treatment is not indicated .

BVOS FOR NEOVASCULARIZATION

BVOS demonstrated that prophylactic scatter photocoagulation can reduce the development of neovascularization and in patients with already existing neovascularization it can reduce the risk of vitreous hemorrhage .

It also states that only in eyes with >5disc diameter of capillary non perfusion areas are at risk of developing neovascularization , about 40% of the eyes with >5 disc diameter of capillary non perfusion develop neovascularization . Out of this 40% , 60% of eyes will go for vitreous hemorrhage . Peripheral sectoral photocoagulation in eyes with large capillary non perfusion areas will reduce neovascularization by 40 to 20% . But if we treat all eyes with sectoral laser photocoagulation prophylactically , 60% of eyes that would never develop neovascularization would receive laser .

Therefore BVOS strongly recommends sectoral laser photocoagulation only after neovascularization is observed , and states that it is as effective as prophylactic sectoral laser photocoagulation in preventing vitreous hemorrhage .

The laser photocoagulation can be applied with argon blue – green laser to achieve medium white burns of size 200 – 500 µm in diameter spaced one burn width apart covering the entire capillary nonperfusion area defined by FFA , not closer than 2 disc diameter from the center of the fovea and extending peripherally at least to the equator .

The complications of laser are infrequent . Laser should not be placed over the intraretinal hemorrhages as it absorbs the laser energy and likely damage the nerve fiber layer rather than laser reaching the RPE and thereby enhancing the development of the preretinal fibrosis .

The side effects of laser treatment are inadvertent foveal burns , loss of peripheral vision , choroidal neovascularzation due to breaks in bruch's membrane , scotomas , therefore patient should be well informed before the initiation of the treatment .

If left untreated 60% of pts who develop neovascularization will go for vitreous hemorrhage leading to prolonged visual disability in some patients . If the hemorrhage is dense B-scan should be done to look for associated tractional retinal detachment . If vitreous hemorrhage does not clear in few months , pars plana vitrectomy with sectoral endolaser photocoagulation can be done .

Other complications like iris neovascularization are rare in BRVO but in diabetic patients with BRVO the risk is increased . Collaterals develop frequently around the blockage site as vein to vein channels and may mimic neovascularization. FFA helps in differentiating collaterals from neovascularization .

Fig 9: Showing fundus picture of a IT BRVO patient , with FFA showing NVE and fundus picture showing sectoral photocoagulation marks in the inferior quadrant .

STEROID TREATMENT

Increased vascular permeability is mediated partly by an increase in VEGF leading to macular edema .

Corticosteroids inhibit the VEGF and its anti – inflammatory property also helps in reducing the macular edema .

Intraocular corticosteroids has many side effects including mainly glaucoma and cataract formation .

SCORE STUDY

It is a standard care vs corticosteroid study for BRVO . It studied the effectiveness of intravitreal triamcinolone acetate (IVTA) for the treatment of macular edema in BRVO . It is a multicenter , randomized controlled study . 411 patients were randomized to receive macular grid laser , 1mg IVTA , 4 mg IVTA . Retreatment was allowed every 4 months for each group , unless the treatment was successful or futile or contraindicated . It is found that there is no significant difference in vision or reduction of macular edema measured by OCT at the end of 12 months between each group . Therefore IVTA is not recommended as first line therapy for macular edema in BRVO . It can only be considered in patients when other modalities fail , especially in pseudophakic eyes .

GENEVA STUDY

The global evaluation of implantable dexamethasone in retinal vein occlusion with macular edema study evaluated a sustained release , biodegradable , dexamethasone intravitreal implant for the treatment of macular edema in CRVO and BRVO . It is studied to evaluate the effectiveness of Ozurdex 0.7mg , Ozurdex 0.35mg , and sham treatments .

Ozurdex is a biodegradable copolymer of poly D,L-lactide-co-glycolide acid (PLGA) containing micronized dexamethasone . It is injected using 23 gauge custom injector through pars plana route . It gradually releases the dexamethasone via Krebs cycle breakdown of the PLGA into lactic acid and glycolic acid and finally into water and carbondioxide .

A statistically significant difference between both ozurdex groups and sham group was seen upto 90days after injection . There was significant improvement in retinal thickness measured by OCT in both ozurdex groups , compared to sham groups .

The only complications that are more in ozurdex groups than sham group are increase in IOP and anterior chamber cells .

The GENEVA study showed that the dexamethasone implant is an alternative treatment to macular grid laser in the appropriate patients . It is FDA approved for this indication .

ANTI – VEGF TREATMENT

Retinal ischemia leads to secretion of VEGF , which inturn leads to increased vascular permeability , vasodilation , migration of endothelial cells , and neovascularization . Increased vascular permeability and and vasodilation leads to macular edema .

Anti – VEGF available at present are Ranibizumab , Bevacizumab , Pegaptanib , Aflibercept .

RANIBIZUMAB :

- A humanized monoclonal antibody fragment , has only a single affinity-matured binding site for VEGF
- Non-selectively binds and inhibits all isoforms of VEGF-A
- Molecular weight 48 kilo daltons (better retinal penetration)
- Usual dosage – 0.5mg/0.05ml , systemic half life – 4hrs

BRAVO STUDY :

The branch retinal vein occlusion study is a prospective , multicenter , randomized controlled study to evaluate the efficacy and safety of ranibizumab and traditional laser in the treatment of macular edema in BRVO .

Patients are grouped into 3 groups , Ranibizumab 0.3mg group , Ranibizumab 0.5mg group and sham group . For the first 6 months monthly injections are given , with rescue laser at 3 months in eligible patients .

It showed that Ranibizumab is superior to the traditional laser treatment for macular edema in BRVO . Therefore the current recommendation is to treat the patients with macular edema from BRVO with 0.5mg Ranibizumab monthly . If treatment fails after 3 consecutive monthly injections of Ranibizumab then traditional grid laser should be performed .

Currently the only Anti – VEGF that is FDA approved for treating macular edema from BRVO is Ranibizumab .

Other Anti – VEGF's are..

BEVACIZUMAB :

- A complete antibody , with two binding sites for VEGF
- Pan blocker of all isoforms of VEGF
- Its use is off label
- Molecular weight 149 kilo daltons
- Cheaper than Aflibercept and Ranibizumab
- It is approximately comparable to Ranibizumab in efficacy and safety , except that some assessments suggested marginally raised serious systemic adverse effects compared to Ranibizumab
- Treatment strategies are similar to Ranibizumab
- Usual dosage – 1.25mg/0.05ml , systemic half life – 20hrs
- It is FDA approved for colorectal cancer , but is used off – label in the eye .

PEGAPTANIB :

- It was the first anti-VEGF agent approved for use in the eye
- It is an aptamer which blocks major angiogenic isoform 165 of VEGF

AFLIBERCEPT :

- A recombinant fusion protein
- Binds to VEGF-A , VEGF-B , PIGF (placental growth factor)
- An induction course of three injections is given at monthly intervals
- Recommended maintenance regimen – one injection every 2 months
- Standard dosage- 2mg/0.05ml

EXPERIMENTAL TREATMENTS

FAVOR STUDY :

A sustained release , non erodible , intravitreal implant of fluocinolone acetonide (Iluvien) is currently under investigation , for use in patients with macular edema from BRVO or CRVO .

This implant is introduced into vitreous cavity with a 25 gauge injector . It releases 0.2 µg /day for about 36 months .

SURGICAL MANAGEMENT :

The majority of the venous lesions in BRVO occur at the arteriovenous crossing site . In a restrospective review of the colour photographs of FA of BRVO patients , kumar and associated found venous narrowing at the crossing site , evidence of downstream hemodynamic changes on angiogram , and presumed thrombi . They also suggested that sheathotomy can relieve the compression and may be effective treatment in BRVO .

Osterloh and Charles presented the first report on sheathotomy for BRVO with significant visual improvement in one case .

Opremcak and Bruce presented the second report on sheathotomy for BRVO with equal or improved visual acuity in 12 out of 15 cases (80%) .

Mester and Dillinger reported 43 cases of BRVO treated with sheathotomy with similar results .

Cahill and colleagues reported 27 cases of BRVO with vitrectomy and sheathotomy without a statistically significant visual improvement .

Since there is evidence that vitreomacular attachment itself may contribute to the development o macular edema in BRVO , Saika and coworkers reported reduction in macular edema and restoration of foveal contour in 10 of 19 eyes after vitrectomy , posterior hyaloids separation , and intraocular gas tamponade .

FOLLOW – UP

The vision threatening complications in BRVO are macular edema , macular ischemia and neovacularization . Treatment is only available for macular edema and neovascularization . Initially patients should be closely followed every 2months to look for these treatable causes of vision loss . Each patient should have a tailored approach of treatment and treated accordingly .

AIM OF THE STUDY

To compare the visual outcome and prognosis in patients with Branch Retinal Vein Occlusion who underwent Early Sectoral Photocoagulation v/s the patients who underwent Sectoral Photocoagulation after development of neovascularization (as suggested in Branch vein occlusion study).

PRIMARY OBJECTIVE

To observe the visual outcome and prognosis in Branch Retinal Vein Occlusion after Early Sectoral Photocoagulation .

SECONDARY OBJECTIVE

To observe the development of complications following Early Sectoral Photocoagulation .

SAMPLE SIZE : Study group : 30 patients

Control group : 30 patients

Total sample size : 60 patients

DURATION : November 2017-September 2018

METHODOLOGY (MATERIALS AND METHODS)

BRVO patients presenting to uvea and retina services will be registered, evaluated and followed up during the study period.

A detailed history of the patient will be taken. Complete general examination with vitals measurement will be performed . Examination of RS , CVS , CNS will be performed .

Ocular examination including best corrected visual acuity (using Snellen's chart) , anterior and posterior segment examination using Slit lamp , Direct Ophthalmoscopy , slit lamp biomicroscopy and 90D, indirect ophthalmoscopy with 20D will be done . Intraocular pressure (Goldmann applanation tonometry) will be measured .

Ancillary tests such as Fundus Fluorescein Angiogram , Optical Coherence Tomography when needed .

Investigations such as Blood Pressure , Fasting Blood Sugar , Post Prandial Blood Sugar , Fasting Lipid Profile , Total Count , Differential Count , Erythrocyte Sedimentation Rate , Haemoglobin Percentage , Peripheral Blood Smear will be done.Patients will be referred to Cardiologist for Echo , Carotid Doppler and Rheumatologist for ANA,RF,cANCA when needed .

In our study 60 patients of BRVO will be divided equally into two groups , a control group (30 patients) and a study group (30 patients) . Informed consent will be obtained from each patient before the study starts .

The patients in control group will be subjected to FFA once the intraretinal haemorrhages subside . The patients with neovascularization will be treated with sectoral photocoagulation and followed up . The patients without neovascularisation will be kept under observation and followed up . If neovascularisation develops on follow up , then sectoral photocoagulation will be done as suggested by BVOS .

The patients for study group will be subjected to FFA after intraretinal haemorrhages subside and the patients without neovascularisation will be included in the study group . Sectoral photocoagulation will be done to all the patients in study group (i.e., before neovascularisation develops) and followed up to look for any development of complications following early sectoral photocoagulation .

INCLUSION CRITERIA

- Branch retinal vein occlusion either Supero-Temporal or Infero- Temporal
- Unilateral or bilateral Branch Retinal Vein Occlusion

EXCLUSION CRITERIA

- Patients with Central Retinal Vein Occlusion/Hemi Retinal Vein Occlusion/Tributary Vein Occlusion
- Patients with other retinal diseases
- Patients treated else where for the same disease

EVALUATION OF THE PATIENT

A detailed history of the patient will be taken. Complete general examination with vitals measurement will be performed . Examination of RS , CVS , CNS will be performed .

Ocular examination including best corrected visual acuity (using Snellen's chart) , anterior and posterior segment examination using Slit lamp , Direct Ophthalmoscopy , slit lamp biomicroscopy and 90D, indirect ophthalmoscopy with 20D will be done . Intraocular pressure (Goldmann applanation tonometry) will be measured .

Ancillary tests such as Fundus Fluorescein Angiogram , Optical Coherence Tomography when needed .

Investigations such as Blood Pressure , Fasting Blood Sugar , Post Prandial Blood Sugar , Fasting Lipid Profile , Total Count , Differential Count , Erythrocyte Sedimentation Rate , Haemoglobin Percentage , Peripheral Blood Smear will be done.Patients will be referred to Cardiologist for Echo , Carotid Doppler and Rheumatologist for ANA,RF,cANCA when needed .

Follow up : 1st week

1st month

3rd month

6th month

As and when needed.

ASSESSMENTS OF PARAMETERS :

- Visual acuity
- Development of new vessels
- Intraocular pressure

MANAGEMENT DONE IN OUR STUDY :

In our study 60 patients of BRVO were divided equally into two groups , a control group (30 patients) and a study group (30 patients) . Informed consent was obtained from each patient before the study started .

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RESULTS AND ANALYSIS

AGE DISTRIBUTION IN CONTROL GROUP

	Control group
Age Distribution	Percent
<40 y	3.33
41 – 50 y	26.67
51 – 60 y	46.67
61 – 70 y	13.33
>70 y	10
Total	100

Table 1: Age distribution in control group .

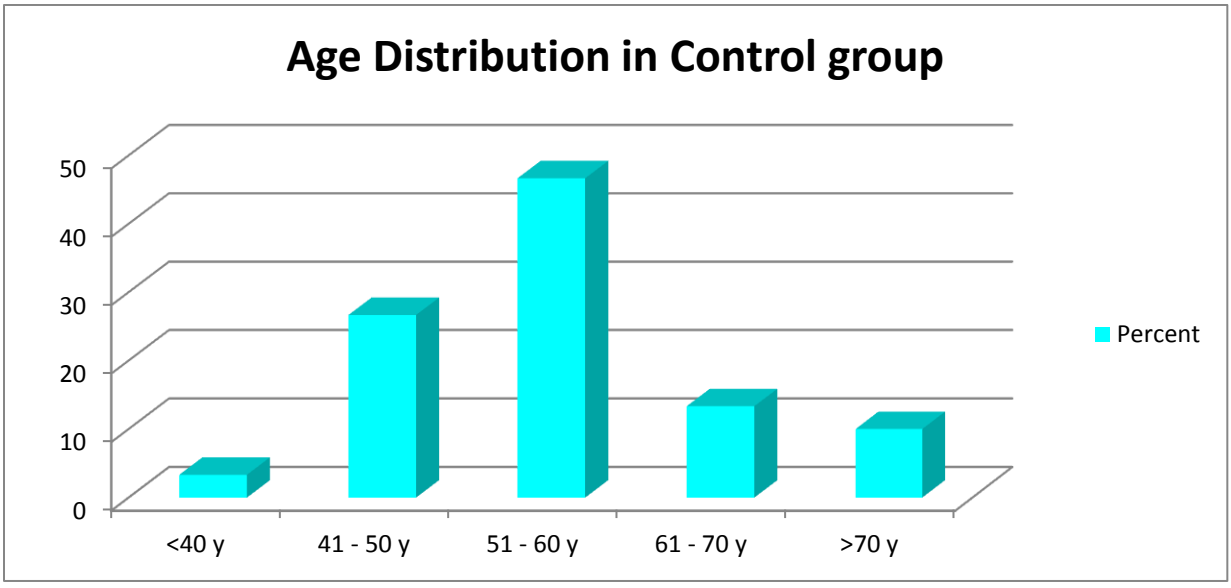


Chart 1: Age distribution in control group .

In control group , maximum number patients are in the age group of 51 – 60 years accounting to 46.67% .

AGE DISTRIBUTION IN STUDY GROUP

Study group	
Age Distribution	Percent
<40 y	6.67
41 – 50 y	33.33
51 – 60 y	43.33
61 – 70 y	13.33
>70 y	3.33
Total	100

Table 2: Age distribution in study group .

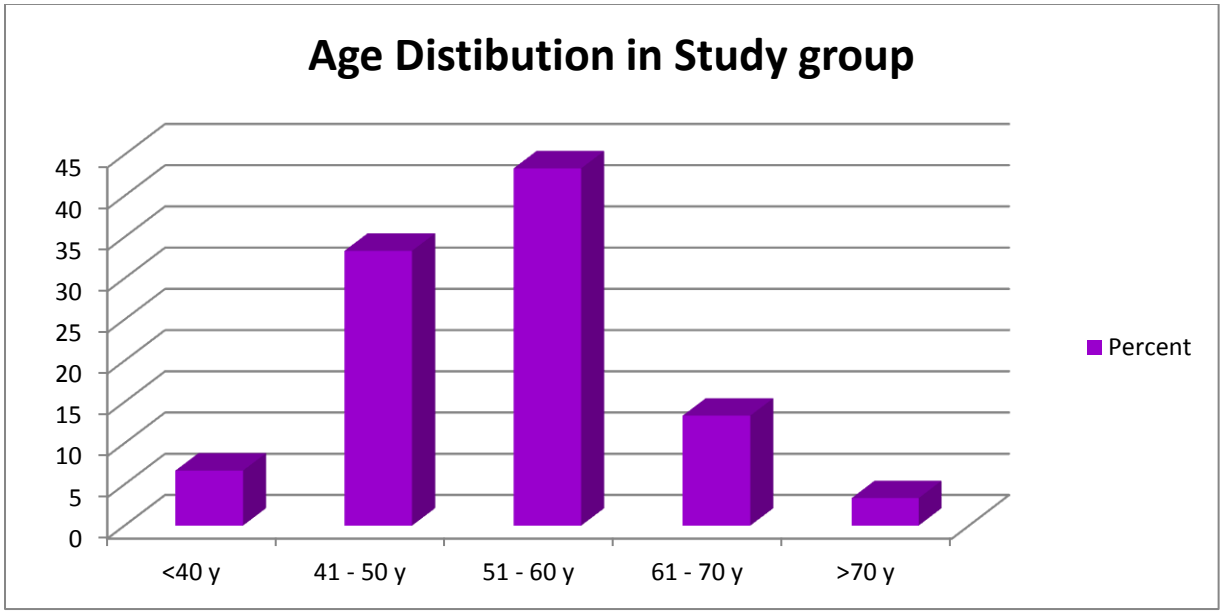


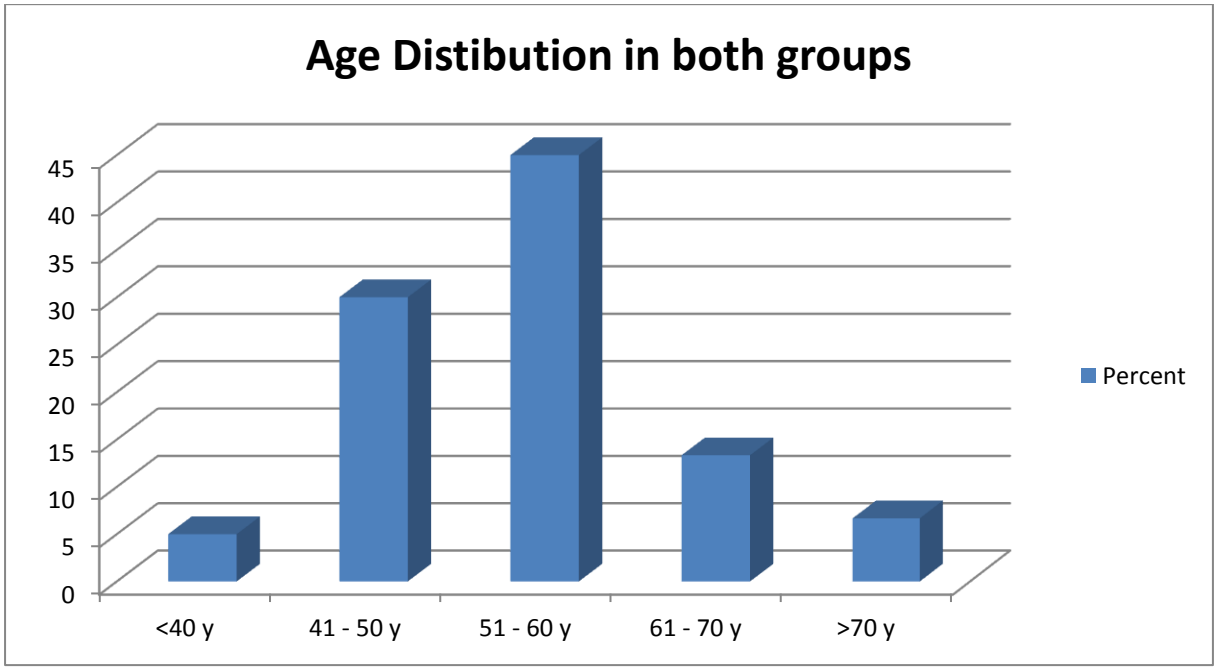
Chart 2: Age distribution in study group .

In study group , maximum number of patients are in the age group of 51 – 60 years , accounting to 43.33% .

AGE DISTRIBUTION IN THE STUDY

Age Distribution	Percent
<40 y	5
41 – 50 y	30
51 – 60 y	45
61 – 70 y	13.33
>70 y	6.67
Total	100

Table 3: Age distribution in both the groups .



Charts 3: Age distribution in both the groups .

The age distribution together in both control and study group , maximum number of patients are in the age group of 51 – 60 years , accounting to 45% .

MEAN AGE DISTRIBUTION IN THE STUDY

	Total		Control		Study		
Variable	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	p value
N	60		30		30		
age	54.15	9.18	55.67	8.86	52.63	9.39	>0.05

Table 4: Mean age of patients included in the study .

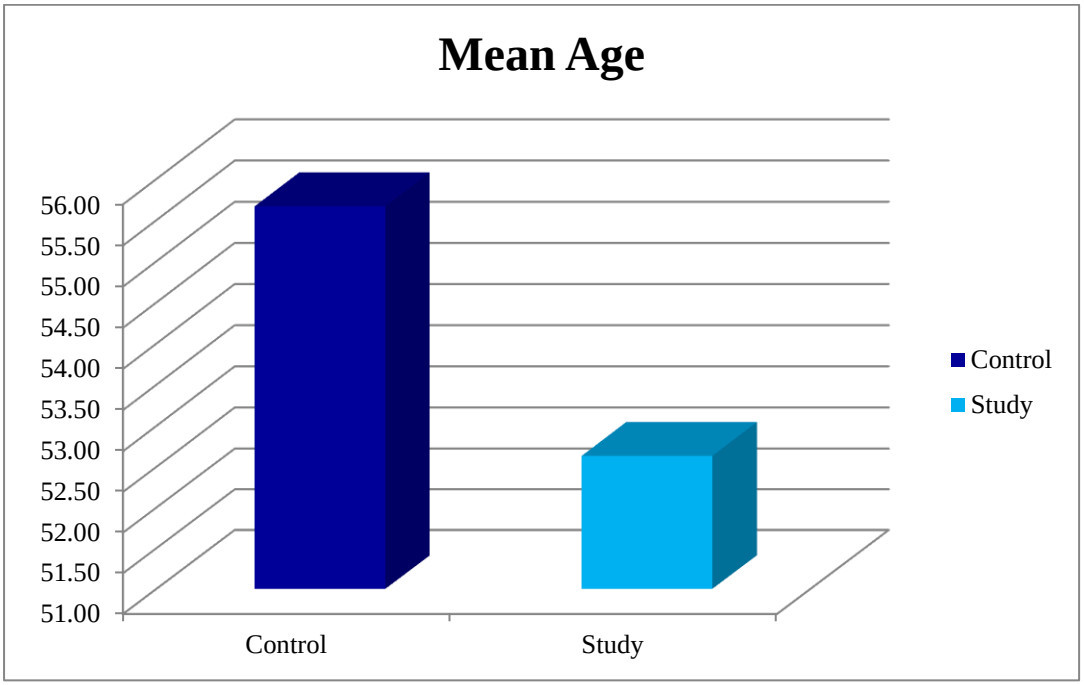


Chart 4: Mean age distribution in control group and study group .

The age distribution in our study showed that the mean age of affected are 54.15 years .

GENDER DISTRIBUTION IN CONTROL GROUP

	Control group	
SEX	Freq.	Percent
F	14	46.67
M	16	53.33
Total	30	100

Table 5: Gender distribution in control group .

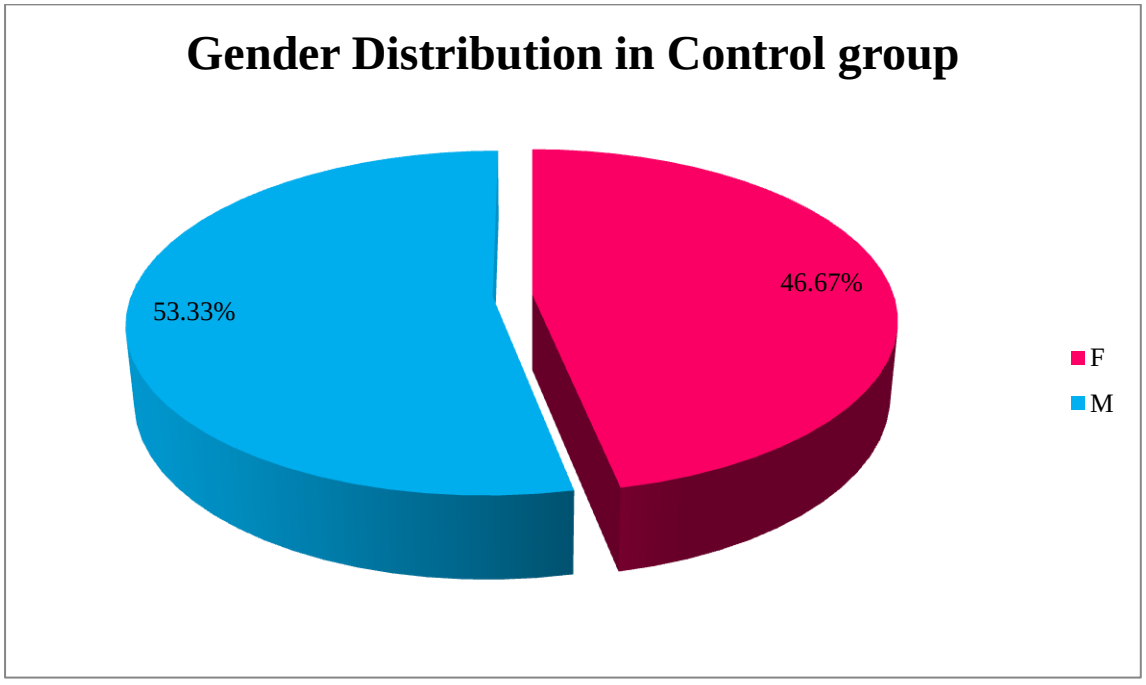


Chart 5: Pie chart showing the gender distribution in control group .

In control group , 53.33% of patients are males and 46.67% of patients are females .

GENDER DISTRIBUTION IN STUDY GROUP

	Study group	
SEX	Freq.	Percent
F	16	53.33
M	14	46.67
Total	30	100

Table 6: Gender distribution in study group .

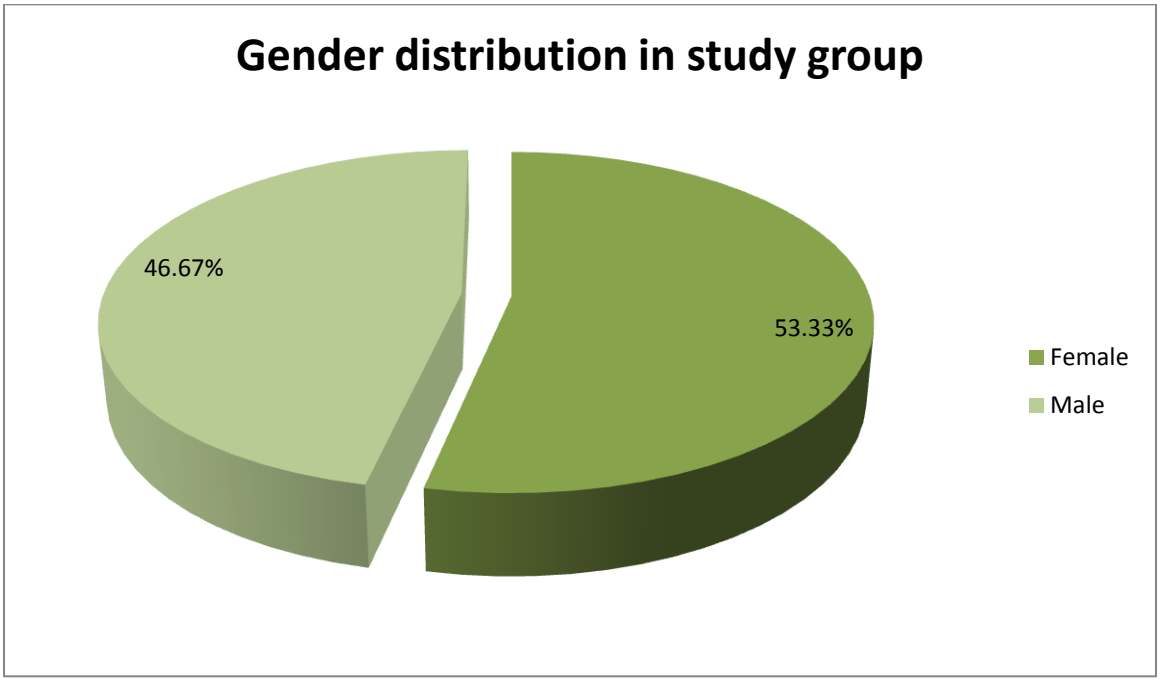


Chart 6: Pie chart showing the gender distribution in study group .

In study group , 46.67 % of patients are Males and 53.33 % patients are females .

GENDER DISTRIBUTION IN THE STUDY

Gender	Frequency	Percentage
Female	30	50
Male	30	50
Total	60	100

Table 7: Gender distribution in this study

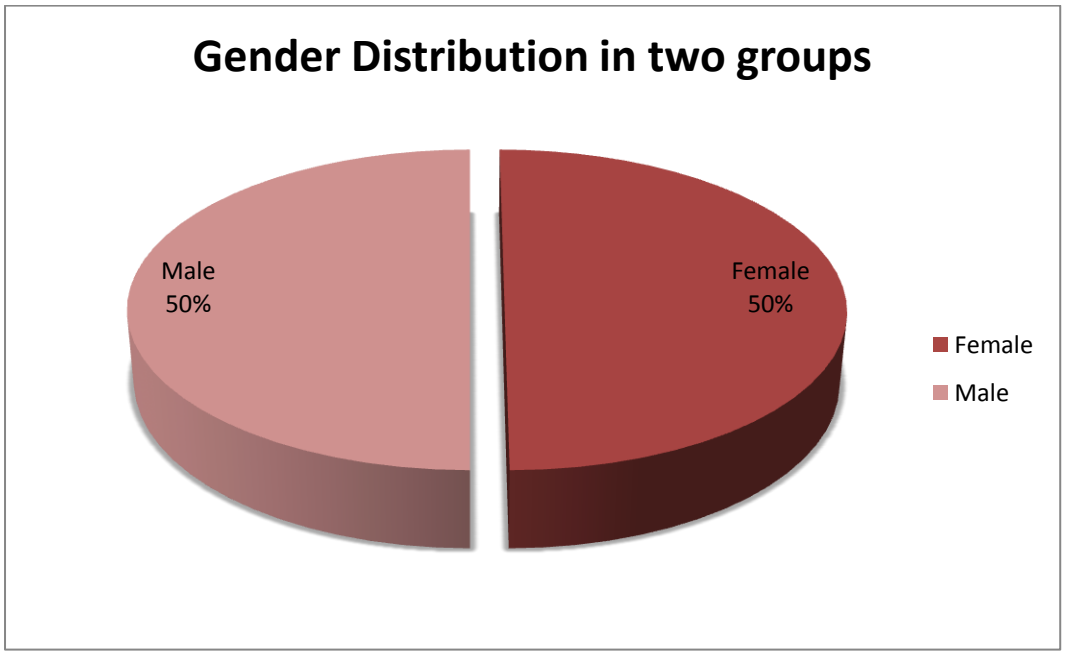


Chart 7: Pie chart showing gender distribution in this study .

In this study , 50% of the patients are males and 50% of the patients are females .

SYSTEMIC ILLNESS ASSOCIATION

	Total		Control group		Study group		
SYSTEMIC ILLNESS	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
HTN	51	85	26	86.66	25	83.33	>0.05
HTN , DM	9	15	4	13.33	5	16.67	
Total	60	100	30	100	30	100	

Table 8: Distribution of systemic illness in control and study group .

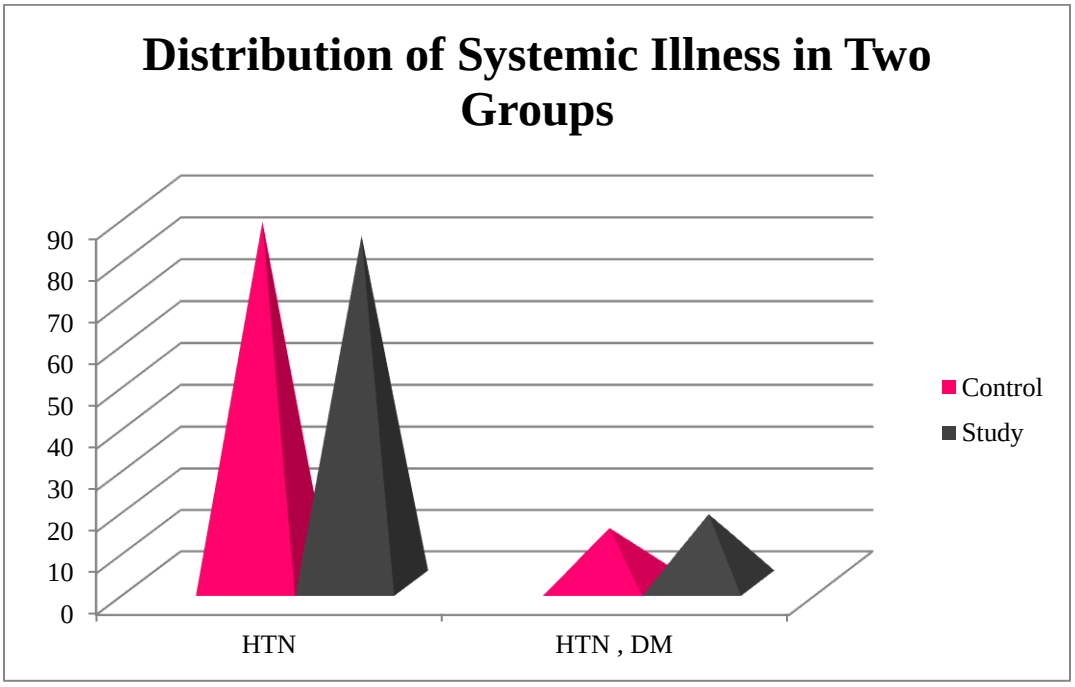


Chart 8: Distribution of systemic illness in control group and study group .

In this study , 85% are hypertensive patients and 15 % are both hypertensive and diabetic patients .

LATERALITY

EYE INV	Total		Control group		Study group		
	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
RE	35	58.33	15	50	20	66.67	>0.05
LE	25	41.67	15	50	10	33.33	
Total	60	100	30	100	30	100	

Table 9: Eye involved in the study .

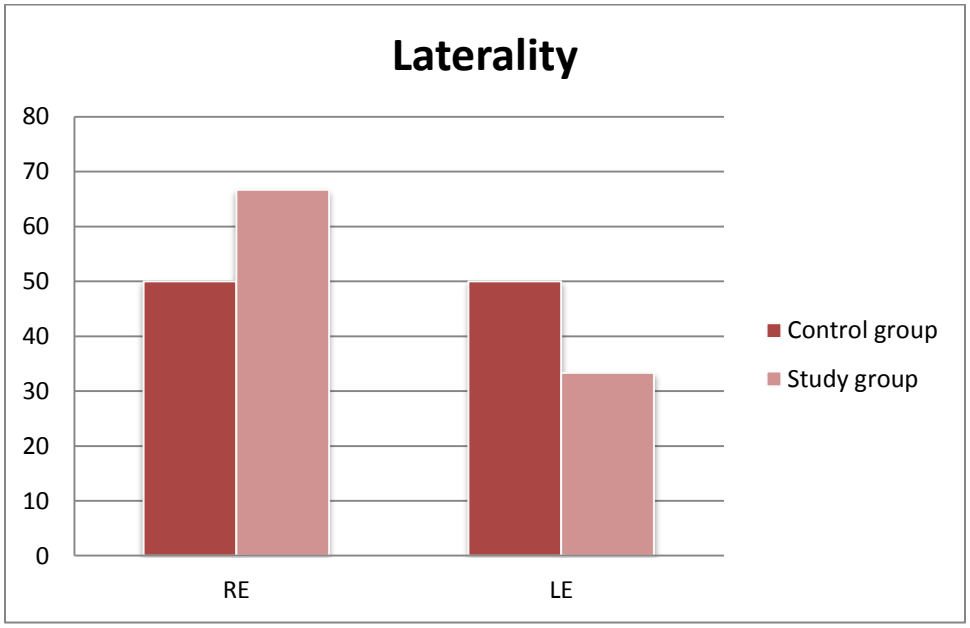


Chart 9: Eye involved in the study .

In this study , right eye is involved 58.33% of the times , and left eye is involved 41.67% of the times .

PRESENTING SYMPTOMS

SYMPTOMS	Total		Control group		Study group		
	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
Defective vision	57	95	27	90	30	100	>0.05
Defective vision, Floaters	3	5	3	10	0	0	
Total	60	100	30	100	30	100	

Table 10: Presenting symptoms of the patients .

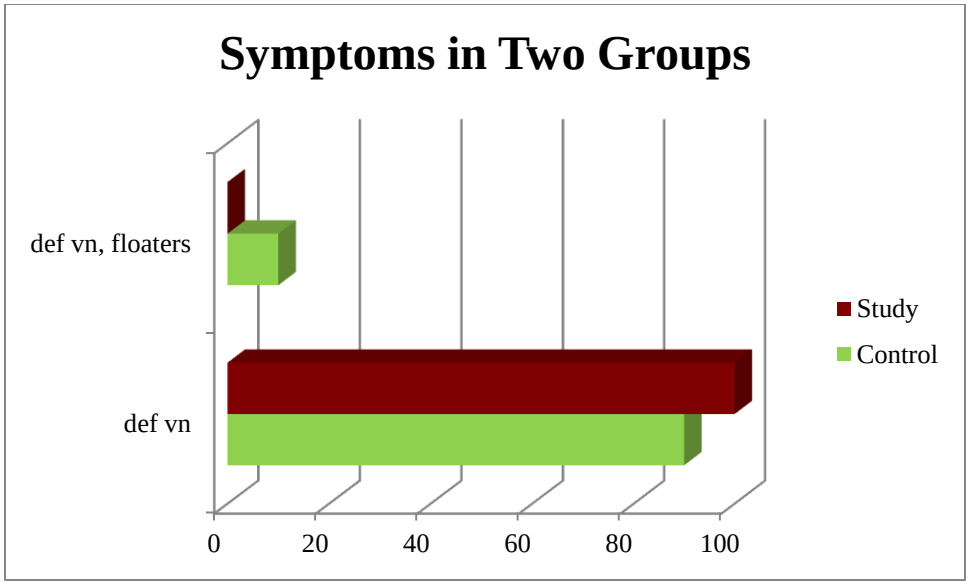


Chart 10: Presenting symptoms of the patients .

In this study , 95% of the patients presented with complaints of defective vision , and 5% presented with complaints of defective vision and floaters .

TYPE OF BRVO

TYPE	Total		Control group		Study group		
	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
ST BRVO	39	65	19	63.33	20	66.67	>0.05
IT BRVO	21	35	11	36.67	10	33.33	
Total	60	100	30	100	30	100	

Table 11: Type of BRVO frequency in this study .

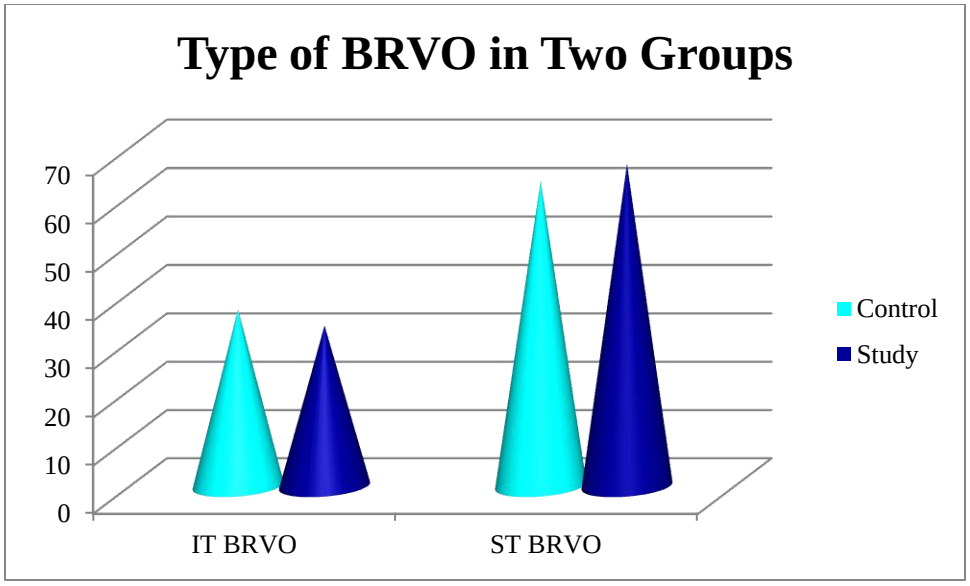


Chart 11: Type of BRVO frequency in this study .

In this study , 65% of the cases are ST BRVO and 35% of the cases are cases of IT BRVO .

PRE AND POST TREATMENT VISION

Variable	Total		Control group		Study group		
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	p value
Affected Eye Vision	0.62	0.61	0.77	0.83	0.47	0.15	>0.05
Post Treatment Vision	0.45	0.44	0.54	0.6	0.36	0.15	>0.05

Table 12: Vision change after treatment in both the groups .

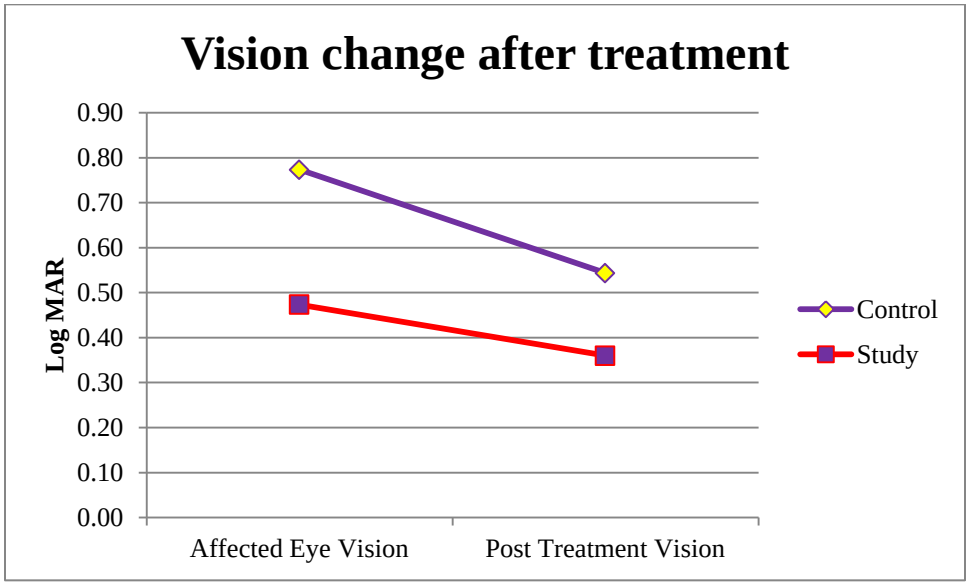


Chart 12: Vision change after treatment .

In our study , the mean visual acuity improvement by snellen's chart in control group is from about 6/36 to 6/18 , and in study group is from about 6/18 to 6/12 . The mean visual acuity improvement of all the patients was 6/24 to 6/12p .

VISION DIFFERENCE

	Total		Control group		Study group		
Variable	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	p value
Vision Difference	0.17	0.32	0.23	0.42	0.11	0.14	>0.05

Table 13: Vision difference between the two groups .

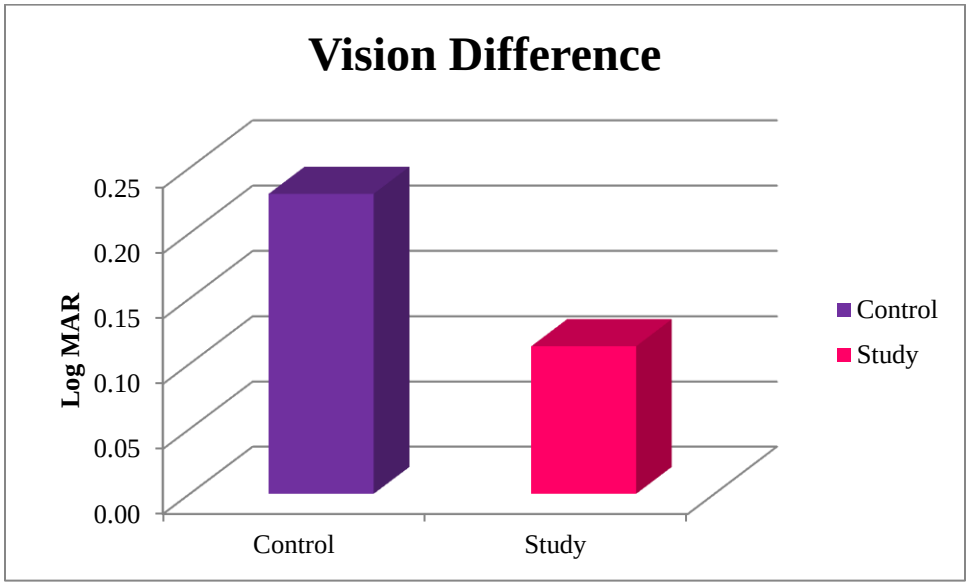


Chart 13: Vision difference between the two groups .

PRE AND POST TREATMENT MEAN IOP

	Mean IOP in mmHG	Control group	Study group
Before Rx	12.5	12.37	12.63
After Rx	12.35	12.33	12.37

Table 14: Mean IOP difference pre and post treatment .

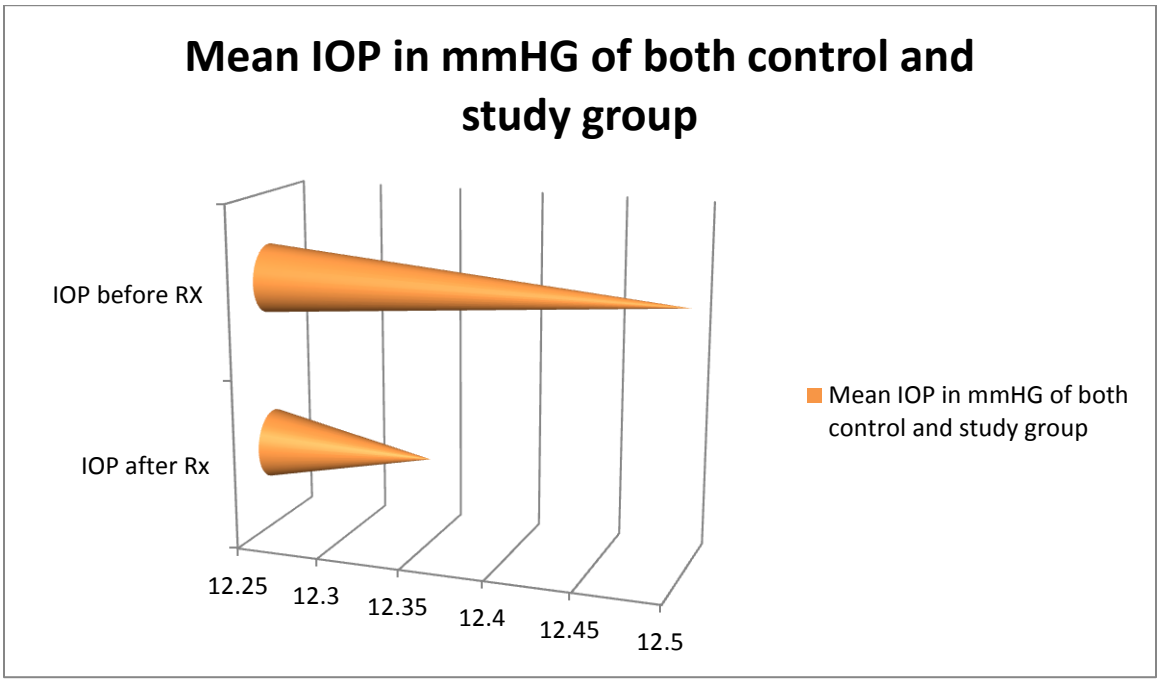


Chart 14: Mean IOP difference in both control and study group pre and post treatment .

In our study , pre and post treatment IOP did not show much variations and was found to be well with in the normal limits .

DISTRIBUTION OF COMPLICATIONS

COMPLICATIONS	Total		Control group		Study group		
	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
NIL	42	70	12	40	30	100	<0.001
NVD	5	8.33	5	16.67	0	0	<0.05
NVD & NVE	2	3.34	2	6.66	0	0	>0.05
NVE	6	10	6	20	0	0	<0.01
VH	5	8.33	5	16.67	0	0	<0.05
Total	60	100	30	100	30	100	

Table 15: Distribution of complications in the study

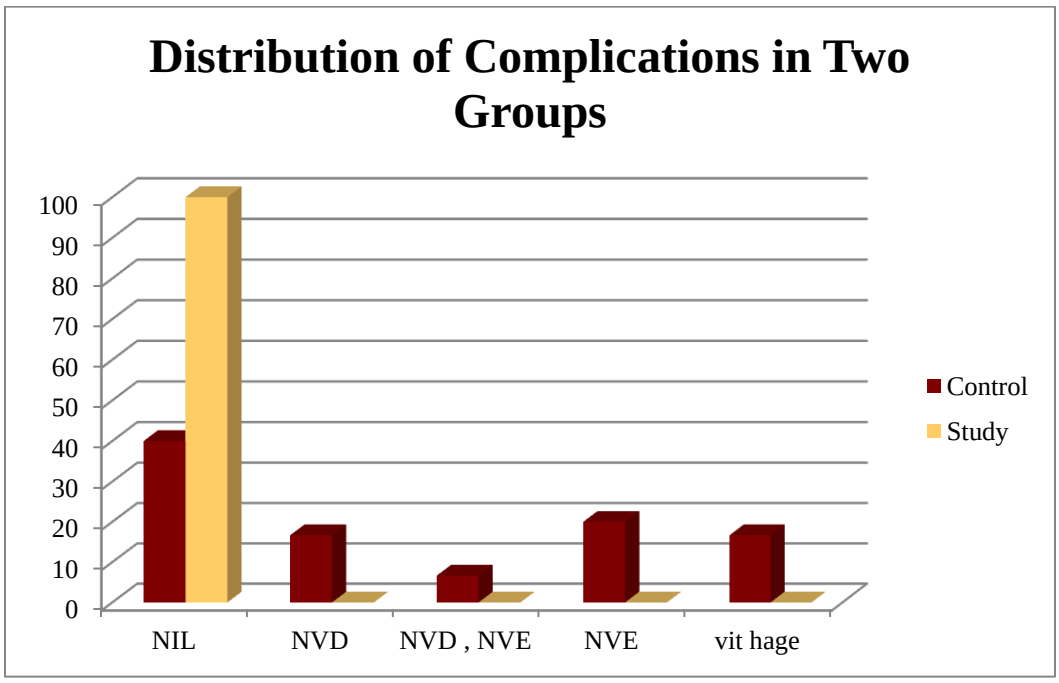


Chart 15: Distribution of complications

In our study , in control group patients , 16.67% of the patients developed NVD , 20% developed NVE , 6.66% developed both NVD and NVE , 16.67% developd vitreous hemorrhage , 40% developed no complications .

In study group , no patients developed NVD or NVE or Vitreous hemorrhage.

MANAGEMENT OF THE AFFECTED EYE

	Total		Control		Study		
MANAGEMENT OF AFFECTED EYE	Freq.	Percent	Freq.	Percent	Freq.	Percent	p value
Observation	12	20	12	40	0	0	<0.001
Sectoral PRP	43	71.67	13	43.33	30	100	<0.001
Sectoral PRP where ever possible	5	8.34	5	16.67	0	0	<0.05
Total	60	100	30	100	30	100	

Table 16: Management of the affected eye

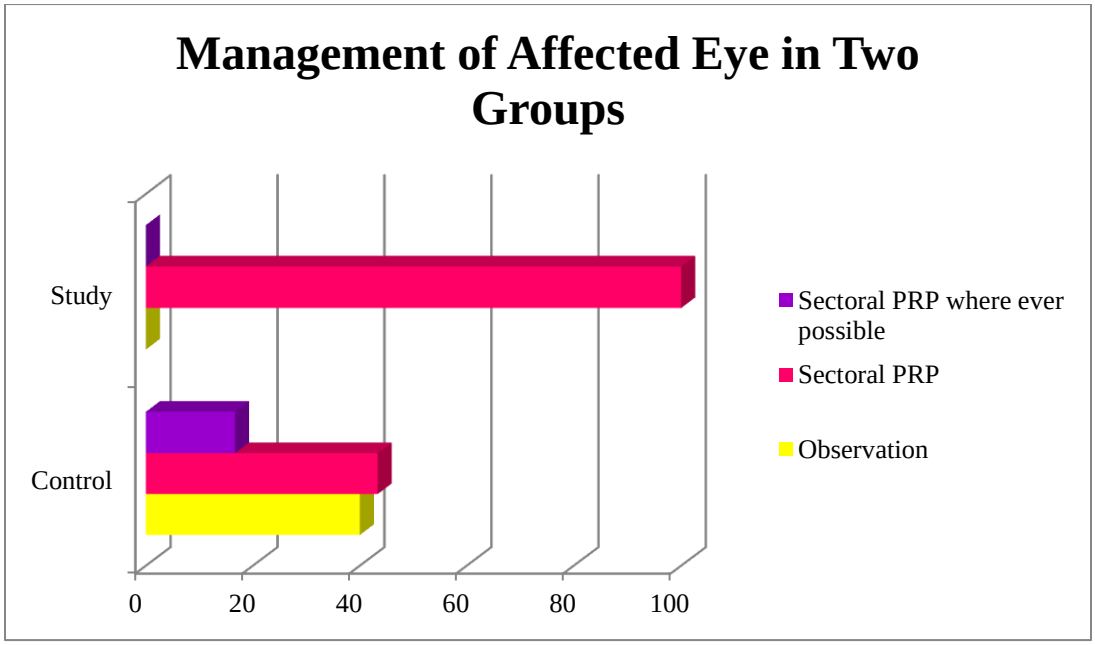


Chart 16: Management of the affected eye

In our study , among control group 40% of the patients are kept under observation , 60% of the patients required sectoral photocoagulation eventually. Among study group , sectoral photocoagulation is done to all the patients .

DISCUSSION

1) Age distribution

In our study , maximum number of patients were in the age group of 50 – 60 years , accounting to about 45% of the patients included in both control group and study group . The mean age in our study is found to be 54.15 years . The Beaver Dam Eye Study showed most patients with in the age group of 60 – 70 years .

2) Gender distribution

In our study , males and females are equally effected accounting to 50% each , which is also noted in The Beaver Dam Eye study .

3) Systemic illness association

In our study , 85% of the patients has hypertension and 15% of the patients has both hypertension and diabetes . The association of BRVO with systemic hypertension is also shown in many prior studies like The Eye Disease Case Control Study Group .

4) Laterality

In our study , all the patients had unilateral involvement .The incidence of BRVO is more in right eye (58.33%) than in left eye (46.67%) in our study . The Beaver Dam Eye study also shows equal incidence of BRVO in both the eyes .

5) Presenting symptoms

In our study , 95% of the patients presented with complaints of defective vision which is sudden and painless . The remaining 5% of the patients experienced floaters along with sudden painless loss of vision .

6) Affected eye vision at presentation

In our study , the mean vision of the affected eye by snellen’s chart at presentation in control group patients is about 6/36 and in study group is about 6/18 . The mean vision of all the patients in the study at presentation is about 6/24 .

7) Distribution of clinical manifestation

In our study , 63.33% of patients in control group has ST BRVO and 36.67% patients had IT BRVO . 66.67% patients in study group had ST BRVO and 33.33% patients had IT BRVO . The mean distribution of ST BRVO and IT BRVO was 65 % and 35% respectively , which is comparable to other studies .

8) Comparison between presenting vision and post treatment vision improvement

In our study , the mean visual acuity improvement by snellen's chart in control group is from about 6/36 to 6/18 , and in study group is from about 6/18 to 6/12 . The mean visual acuity improvement of all the patients was 6/24 to 6/12p . The over all improvement in vision is one or two lines by snellen's chart .

9) Comparison between presenting IOP and post treatment IOP

In our study , the mean IOP in control group before management is 12.37 mmhg and post management is 12.33mmhg . The mean IOP in study group before management is 12.63mmhg and after management is 12.37mmhg . The mean pre management and post management IOP change is from 12.5mmhg to 12.35mmhg . No significant IOP changes are noted before and after management of BRVO .

10) Development of complications

In our study , in control group patients , 16.67% of the patients developed NVD , 20% developed NVE , 6.66% developed both NVD and NVE , 16.67% developd vitreous hemorrhage , 40% developed no complications .

In study group , no patients developed NVD or NVE or Vitreous hemorrhage .

11) Safety and efficacy of early sectoral photocoagulation

Our study shows that early sectoral photocoagulation is a very safe and effective way to prevent complications like NVD and NVE which leads to Vitreous hemorrhage and maintain the vision .

CONCLUSION

1) The demographic pattern of our study showed most patients in the age group of 50 – 60 years , with mean age of 54.15 years .
2) Males and females are equally affected in our study .
3) All the patients in our study has Hypertension as a known systemic illness , which is a proven risk factor for BRVO .
4) Our study has shown right eye involvement more common than left eye .
5) The commonest symptom in all the patients is defective vision , which is sudden in onset and painless . Few patients with vitreous hemorrhage experienced floaters.
6) The commonest type of BRVO in our study is ST BRVO .
7) The mean visual improvement difference between pre and post treatment in our study is 1 or 2 lines by snellen's chart .
8) In our study , though 12 (40%)out of 30 patients included in the control group did not required any intervention , 13 (43.33%) patients developed NVD or NVE or both and 5 (16.67%) patients developed vitreous hemorrhage even when kept under regular follow up. But among our study group patients , 30 (100%)out of 30 patients did not develop any complications .

9) Therefore in our population especially where the patients compliance to follow up is poor , leading to complications like neovascularization , vitreous hemorrhage with grossly reduced vision for a prolonged period or develop sequelae like tractional retinal detachment or neovascular glaucoma , can be effectively avoided by doing an early sectoral photocoagulation .

10) Therefore early sectoral photocoagulation in BRVO patients is a safe and effective way to prevent complications like NVD , NVE and Vitreous hemorrhage in our population with poor compliance to follow up .

FUNDUS PHOTOGRAPHS

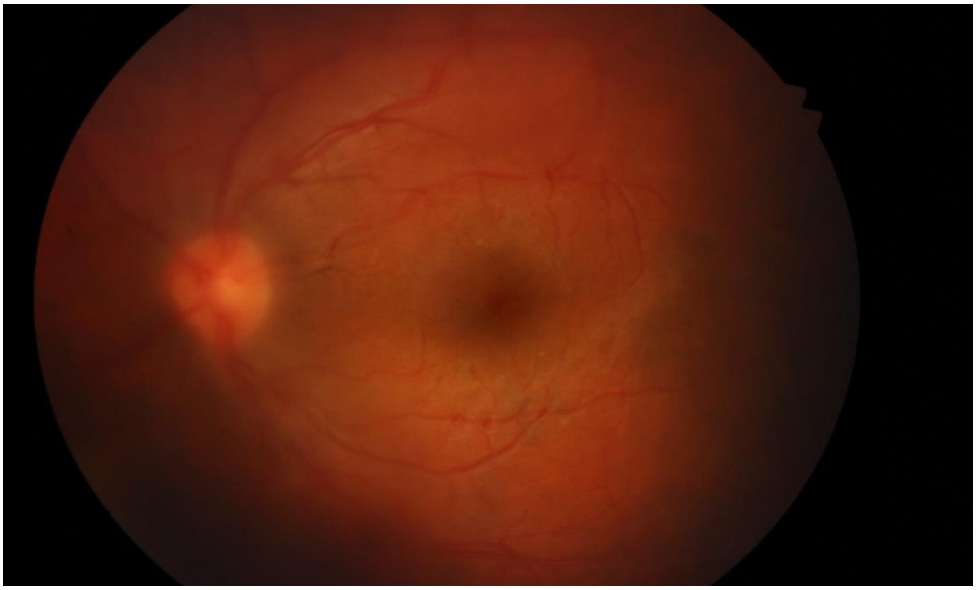


Fig 10a : A fundus photograph of left eye showing IT BRVO with vitreous hemorrhage .

Fig 10b: A FFA picture of the above patient showing the blocked fluorescence due to vitreous hemorrhage .

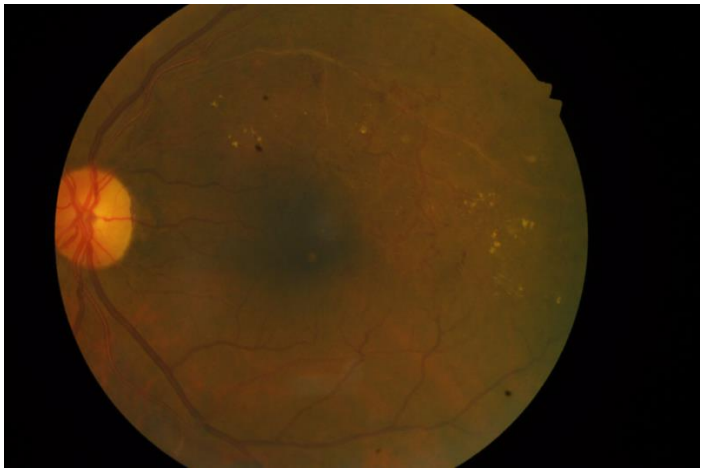


Fig 11a: A fundus photograph showing ST BRVO .

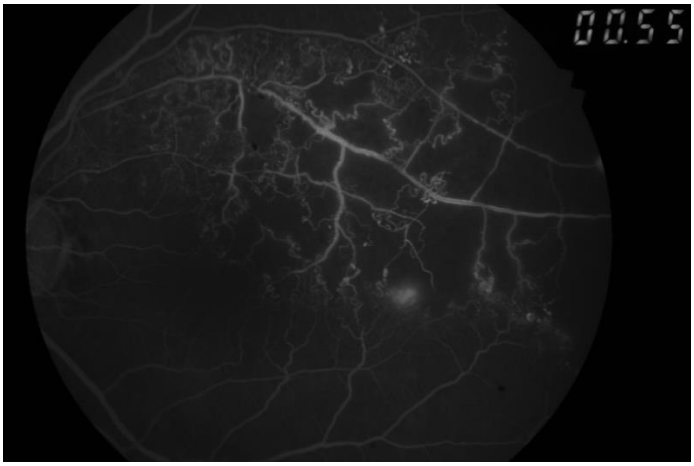


Fig 11b:A FFA picture of the above patient showing NVE following ST BRVO.

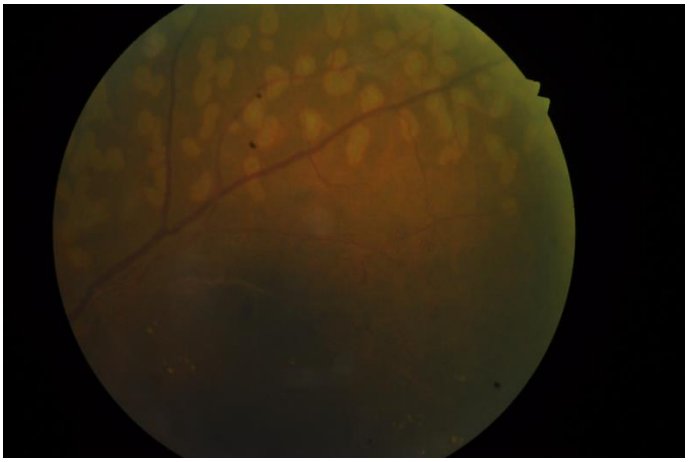


Fig 11c: A fundus photograph of the above patient showing fresh sectoral photocoagulation marks .

Fig 13a: A fundus photograph of left eye showing ST BRVO

Fig 13b: FFA picture of the above patient showing no filling of the affected vessel with CNP areas and few collaterals .

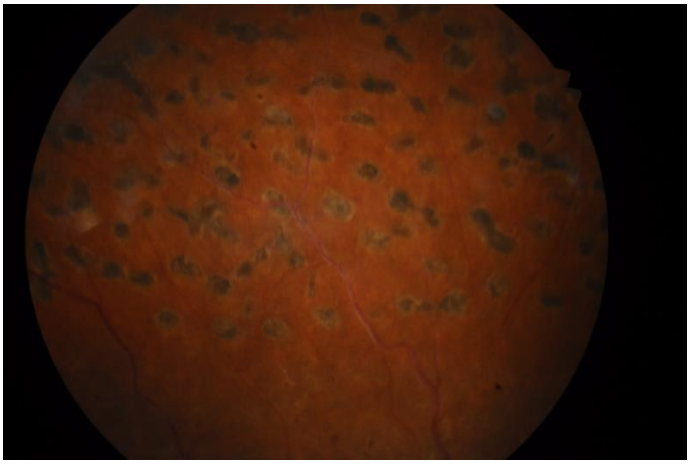


Fig 13c: A fundus photograph of the above patient showing sectoral photocoagulation given before development of neovascularization .

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PROFORMA

NAME :

AGE/SEX :

ADDRESS:

O.P NO :

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

H/O defective vision , duration

PAST HOSTORY:

H/O similar symptoms in the past

H/O diabetes , hypertension , renal failure , hyperlipidemia, cardiovascular disorders

H/O previous ocular surgery.

FAMILYHISTORY

H/O diabetes, hypertension

TREATMENT HISTORY

Medical treatment – Insulin/OHA/anti hypertensive medications

Surgical/laser treatment for the current illness

GENERAL EXAMINATION

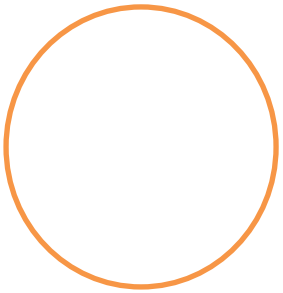
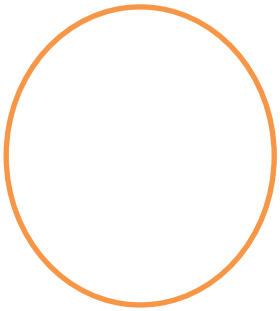
Built

Nourishment

Vitals-pulse, temperature, bloodpressure, respiratory rate

OCULAR EXAMINATION

RIGHT EYE	EXAMINATION	LEFT EYE
	Visual acuity	
	Eyelids	
	Extraocular movements	
	Conjunctiva	
	Cornea	
	Anterior chamber	
	Iris	

	Pupil	
	Lens	
	Fundus examination	
	Intraocular pressure	

OTHER SYSTEMS

CNS,CVS, RS

OTHER CONSULTATION

General Physician and cardiologist Opinion

PROVISIONAL DIAGNOSIS

INVESTIGATIONS

Investigations such as Blood Pressure , Fasting Blood Sugar , Post Prandial Blood Sugar , Fasting Lipid Profile , Total Count , Differential Count , Erythrocyte Sedimentation Rate , Haemoglobin Percentage , Peripheral Blood Smear will be done.Patients will be referred to Cardiologist for Echo , Carotid Doppler and Rheumatologist for ANA,RF,cANCA when needed.

Fundus Fluorescein Angiography.

Optical Coherence Tomography when needed.

KEY TO MASTER CHART

M – Male

F – Female

RE – Right eye

LE – Left eye

NVD – Neovascularization of disc

NVE – Neovascularization else where

VH – Vitreous hemorrhage

HM+ - Hand movement present

CFCF – Counting fingers close to face

DV – Defective vision

NIP – Not improving with pin hole

FL – Floaters

ST BRVO – Supero – temporal branch retinal vein occlusion

IT BRVO – Infero – temporal branch retinal vein occlusion

SP – Sectoral Photocoagulation

OB – Observation

Mx – Management

AFF EYE – Affected eye

CONTROL GROUP

S NO	NAME	SEX	AGE	SYSTEMIC ILLNESS	EYE INV	SYMPTOMS	TYPE	BCVA RE	BCVA LE	LOGMAR OF	IOP in mm HG		COMPLICATIONS	Mx OF AFF EYE	BCVA RE	BCVA LE	LOGMAR	IOP POST Mx	
										AFF EYE	RE	LE			POST Mx	POST Mx	POST Mx	RE	LE
1	Shanthi	F	41	HTN	RE	DV	ST BRVO	6/9p	6/6p	0.2	15	13	NVE	SP	6/9p	6/6p	0.2	15	12
2	Mohana gandhi	M	57	HTN	RE	DV	ST BRVO	6/12p	6/6p	0.3	12	12	NIL	OB	6/9p	6/6p	0.2	11	12
3	Gnanamani	F	75	HTN , DM	LE	DV	ST BRVO	6/9p	6/18P	0.5	10	12	NVD	SP	6/9p	6/18p	0.5	12	12
4	Valli	F	54	HTN	LE	DV	ST BRVO	6/6p	6/18p	0.5	10	10	NIL	OB	6/6p	6/12P	0.3	11	12
5	Ethiraj	M	61	HTN	LE	DV	IT BRVO	6/12p	6/18P	0.5	12	12	NIL	OB	6/12p	6/18P	0.5	13	13
6	Lakshmi	F	47	HTN	RE	DV	IT BRVO	6/12p	6/6p	0.3	12	13	NVE	SP	6/9p	6/6p	0.2	12	13
7	Rajendran	M	58	HTN	LE	DV , FL	ST BRVO	6/9p	HM+	3	13	13	VH	SP where ever possible	6/9p	CFCF	2	15	13
8	Selvaraj	M	48	HTN	LE	DV	ST BRVO	6/6p	6/9P	0.2	14	14	NIL	OB	6/6p	6/6P	0	12	14
9	Balakrishnan	M	52	HTN	RE	DV	ST BRVO	6/18P	6/6p	0.5	12	15	NIL	OB	6/12P	6/6p	0.3	12	13
10	Rasitha	F	65	HTN	RE	DV	IT BRVO	6/24P	6/9p	0.6	12	12	NVD	SP	6/24P	6/9p	0.6	15	12
11	Rajan	M	54	HTN	RE	DV	IT BRVO	6/18P	6/6p	0.5	14	13	NVE	SP where ever possible	6/18P	6/6p	0.5	10	12
12	Velumurugan	M	40	HTN	LE	DV	IT BRVO	6/6p	6/18P	0.5	11	10	NIL	OB	6/6p	6/6P	0	10	11
13	Nasima	F	50	HTN	LE	DV	ST BRVO	6/6p	CFCF	2	12	11	VH	SP where ever possible	6/6p	6/60NIP	1	13	10
14	Kuppusamy	M	63	HTN	LE	DV	ST BRVO	6/12p	6/24P	0.6	13	13	NVE	SP	6/12p	6/18p	0.5	14	13
15	Selvi	F	54	HTN , DM	RE	DV	IT BRVO	6/24P	6/9p	0.6	15	14	NVD , NVE	SP	6/12P	6/9p	0.3	13	12
16	Nagamani	F	57	HTN	RE	DV , FL	ST BRVO	HM+	6/9p	3	12	11	VH	SP where ever possible	HM+	6/9p	3	12	11
17	Deva	M	50	HTN	LE	DV	ST BRVO	6/6p	6/9P	0.2	10	10	NIL	OB	6/6p	6/9P	0.2	10	11
18	Tangam	M	49	HTN	RE	DV	ST BRVO	6/12P	6/6p	0.3	15	15	NIL	OB	6/12P	6/6p	0.3	15	13
19	Mangayar karasi	F	57	HTN , DM	LE	DV	ST BRVO	6/9p	6/18P	0.5	12	12	NVE	SP	6/9p	6/18P	0.5	13	12
20	Pichandi	M	42	HTN	RE	DV , FL	ST BRVO	HM+	6/6p	3	12	13	VH	SP where ever possible	6/60NIP	6/6p	1	14	13
21	Subhapradha	F	75	HTN	RE	DV	ST BRVO	6/24p	6/12p	0.6	10	10	NVD	SP	6/18p	6/12p	0.5	13	10
22	Sevariyammal	F	55	HTN	LE	DV	IT BRVO	6/6p	6/36p	0.8	15	13	NIL	OB	6/6p	6/18P	0.5	15	13
23	Banu	F	65	HTN	LE	DV	IT BRVO	6/18p	6/24p	0.6	12	12	NVE	SP	6/18p	6/24P	0.6	12	11
24	Anandhakrishnan	M	56	HTN	LE	DV	IT BRVO	6/9p	6/18p	0.5	12	10	NVD , NVE	SP	6/9p	6/18p	0.5	10	11
25	Subramani	M	60	HTN	RE	DV	IT BRVO	6/60NIP	6/9p	1	14	11	VH	SP where ever possible	6/36p	6/9p	0.8	12	12
26	Govindhan	M	72	HTN	RE	DV	IT BRVO	6/36p	6/12p	0.8	15	13	NVD	SP	6/18p	6/12p	0.5	12	13
27	Prabhu	M	58	HTN , DM	LE	DV	ST BRVO	6/12p	6/9p	0.3	13	13	NIL	OB	6/12p	6/9P	0.3	11	13
28	Komalavalli	F	54	HTN	RE	DV	ST BRVO	6/12p	6/6p	0.3	13	12	NVD	SP	6/9p	6/6p	0.2	12	12
29	Alphones	F	50	HTN	RE	DV	ST BRVO	6/9p	6/6p	0.2	11	10	NIL	OB	6/6p	6/6p	0	10	11
30	Azhagu	M	51	HTN	LE	DV	ST BRVO	6/6p	6/12p	0.3	15	12	NIL	OB	6/6p	6/12P	0.3	11	13

STUDY GROUP

S NO	NAME	SEX	AGE	SYSTEMIC ILLNESS	EYE INV	SYMPTOMS	TYPE	BCVA RE	BCVA LE	LOGMAR OF	IOP in mm HG		Mx OF	BCVA RE	BCVA LE	LOGMAR	IOP POST Mx		COMPLICATIONS
										AFF EYE	RE	LE	AFF EYE	POST Mx	POST Mx	POST Mx	RE	LE	
1	Manivannan	M	52	HTN	RE	DV	ST BRVO	6/18p	6/9p	0.5	10	11	SP	6/18p	6/9p	0.5	10	10	NIL
2	Rajendran	M	48	HTN	RE	DV	ST BRVO	6/24p	6/6p	0.6	12	12	SP	6/18p	6/6p	0.5	10	12	NIL
3	Shaik nazeer	F	55	HTN , DM	LE	DV	ST BRVO	6/6p	6/12p	0.3	12	12	SP	6/6p	6/9p	0.2	12	12	NIL
4	Vijaya	F	38	HTN	RE	DV	ST BRVO	6/18p	6/6p	0.5	10	11	SP	6/12p	6/6p	0.3	10	10	NIL
5	Moorthy	M	70	HTN	RE	DV	IT BRVO	6/24p	6/12p	0.6	14	12	SP	6/18p	6/12P	0.5	12	13	NIL
6	Egambaram	M	45	HTN , DM	RE	DV	ST BRVO	6/24P	6/6P	0.6	15	15	SP	6/12P	6/6p	0.3	16	16	NIL
7	Vanammal	F	53	HTN	LE	DV	ST BRVO	6/6p	6/24P	0.6	15	13	SP	6/6p	6/9P	0.2	15	13	NIL
8	Marimuthu	M	55	HTN	RE	DV	IT BRVO	6/9P	6/6p	0.2	12	12	SP	6/9P	6/6p	0.2	12	10	NIL
9	Devaraj	M	65	HTN	LE	DV	IT BRVO	6/12p	6/24P	0.6	10	11	SP	6/12P	6/24P	0.6	10	12	NIL
10	Kamala	F	56	HTN	LE	DV	ST BRVO	6/6p	6/18P	0.5	12	10	SP	6/6p	6/9P	0.2	12	12	NIL
11	Sundari	F	60	HTN	RE	DV	IT BRVO	6/36P	6/9p	0.8	14	12	SP	6/18P	6/9p	0.5	14	15	NIL
12	Madhavan	M	75	HTN	RE	DV	ST BRVO	6/12P	6/9p	0.3	10	12	SP	6/9P	6/9p	0.2	10	11	NIL
13	Kabila	F	52	HTN , DM	RE	DV	ST BRVO	6/24P	6/6p	0.6	12	12	SP	6/18P	6/6p	0.5	12	11	NIL
14	Chandrika	F	58	HTN	LE	DV	IT BRVO	6/6p	6/12P	0.3	14	15	SP	6/6p	6/9P	0.2	14	14	NIL
15	Krishnaveni	F	50	HTN	LE	DV	ST BRVO	6/6p	6/12P	0.3	16	14	SP	6/6p	6/9p	0.2	14	12	NIL
16	Dhasarath	M	44	HTN	RE	DV	ST BRVO	6/18p	6/6p	0.5	12	10	SP	6/18p	6/6p	0.5	14	12	NIL
17	Vasuki	F	42	HTN	RE	DV	ST BRVO	6/9P	6/6p	0.2	16	12	SP	6/9p	6/6p	0.2	16	12	NIL
18	Varalakshmi	F	49	HTN , DM	RE	DV	ST BRVO	6/24p	6/6p	0.6	12	10	SP	6/24p	6/6p	0.6	12	10	NIL
19	Shankar	M	51	HTN	LE	DV	ST BRVO	6/6p	6/18p	0.5	12	12	SP	6/6p	6/9p	0.2	12	10	NIL
20	Jayalakshmi	F	44	HTN	RE	DV	ST BRVO	6/24P	6/6p	0.6	10	11	SP	6/18P	6/6p	0.5	10	10	NIL
21	Selvam	M	51	HTN	RE	DV	IT BRVO	6/18p	6/6p	0.5	14	12	SP	6/18p	6/6p	0.5	14	12	NIL
22	Balaji	M	34	HTN	RE	DV	IT BRVO	6/12p	6/6p	0.3	10	10	SP	6/9p	6/6p	0.2	12	11	NIL
23	Santhy	F	43	HTN	LE	DV	ST BRVO	6/6p	6/24p	0.6	12	10	SP	6/6p	6/12p	0.3	15	12	NIL
24	Kumari	F	64	HTN	RE	DV	IT BRVO	6/24p	6/12p	0.6	16	14	SP	6/18p	6/12p	0.5	18	15	NIL
25	Pasupathi	M	59	HTN	RE	DV	ST BRVO	6/18p	6/9p	0.5	14	15	SP	6/18p	6/9p	0.5	12	13	NIL
26	Karunakaran	M	58	HTN	LE	DV	IT BRVO	6/9p	6/12p	0.3	12	12	SP	6/9p	6/9p	0.2	10	12	NIL
27	Ramani	F	47	HTN	LE	DV	ST BRVO	6/6p	6/12p	0.3	16	15	SP	6/6p	6/12p	0.3	14	12	NIL
28	Malathi	F	65	HTN , DM	RE	DV	IT BRVO	6/24P	6/12p	0.6	16	13	SP	6/12p	6/12p	0.3	12	15	NIL
29	Papammal	F	43	HTN	RE	DV	ST BRVO	6/18p	6/6p	0.5	12	10	SP	6/12p	6/6p	0.3	10	12	NIL
30	Babu	M	53	HTN	RE	DV	ST BRVO	6/24p	6/6p	0.3	14	13	SP	6/24p	6/6p	0.6	14	12	NIL